Appendix 4
Human Health

TABLE OF CONTENTS

1,3-Butadiene **716**
Acrolein **722**
Airborne Pathogens **729**
Arsenic **738**
Benzene **753**
Cadmium **759**
Carbon monoxide (CO) **763**
Chromium **770**
Cryptosporidium **779**
Dioxins/Furans **789**
Disinfection By-products **796**
Endocrine Disruptors **804**
Extremely Low Frequency and Electromagnetic Fields (ELF/EMF) **808**
Formaldehyde **810**
Greenhouse Gases **816**
Hantavirus **824**
Indoor Asthma Inducers **830**
Indoor Microbial Air Pollution **837**
Lead **841**
Legionella **850**
Lyme Disease **860**
Mercury **865**
Methyl tertiary butyl ether (MTBE) **876**
Nickel **881**
Nitrogen Pollution (Water) **889**
Nitrogen Oxides (NOx) **896**
Noise Pollution **902**
Ozone (Ground Level) **908**
Particulate Matter (PM) **914**
Pesticides-Food **926**
Pesticides-Indoor **936**
Pesticides - Outdoor **946**
Pesticides -Water **957**
Pfiesteria **967**
Polychlorinated biphenyls (PCBs) **973**
Polycyclic Aromatic Hydrocarbons (PAHs) **987**
Radionuclides **996**
Radium **1005**
Radon **1017**
Secondhand Tobacco Smoke (STS) **1024**
Sulfur oxides (SOx)/Sulfates **1033**
Ultraviolet (UV) Radiation **1038**
Volatile Organic Compounds (VOCs) – Carcinogenic **1046**
Volatile Organic Compounds (VOCs) – Non- carcinogenic **1054**

Waterborne Pathogens **1060**
West Nile Virus **1075**

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**1,3-Butadiene**
Description of stressor (including etiology)	1,3-butadiene, CH_2=CH-CH=CH_2, is a volatile, colorless, flammable gas, with a characteristic, unpleasant to neutral odor.[1] It is produced intentionally and also is a byproduct of incomplete combustion of fossil fuels and biomass. Most of the releases to the environment are from motor vehicle exhaust.[2] About 75% of the intentional production is used to make synthetic rubber, with the remainder used to make plastics including acrylics.[3] Although 1,3-butadiene is soluble in water (solubility = 735 mg/L at 25^o C), its relatively high vapor pressure causes it to readily partition into the gas phase.[4] Thus, releases to soil or water will result in most of the material transferring to the atmosphere. It has a relatively low octanol/water partition coefficient, and is not expected to bioconcentrate.[5] The chemical's half-life in the atmosphere is on the order of one half to several hours.[6,7] The presence of sunlight hastens atmospheric degradation, so that highest concentrations are typically found in the early morning hours.[8] Exposure occurs primarily through breathing contaminated air. High exposures can occur in industrial settings.
stressor-specific impacts considered including key impacts	Breathing very high levels of 1,3-butadiene for even short periods can cause central nervous system damage, blurred vision, nausea, fatigue, headache, decreased blood pressure and pulse rate, and unconsciousness. Breathing lower levels may cause irritation of eyes, nose, and throat. Animal studies have shown that inhalation exposure can increase the number of birth defects, and cause kidney and liver disease and damaged lungs.[9] 1,3-butadiene has recently been upgraded to the status of known human carcinogen by the U.S. Department of Health and Human Services.[10] Epidemiological studies have reported a possible association between 1,3-butadiene exposure and cardiovascular diseases.[11] Animal studies have reported developmental effects, such as skeletal abnormalities and decreased fetal weights, and reproductive effects, including an increased incidence of ovarian atrophy and testicular atrophy, from inhalation exposure.[12] Animal studies have reported tumors at a variety of sites from inhalation exposure. EPA has classified 1,3-butadiene as a Group B2 probable human carcinogen of medium carcinogenic hazard.[13]

Exposure Assessment	
Exposure routes and pathways considered **(include indoor air as appropriate)**	Primary exposure is from breathing contaminated air.
Population(s)/ecosystem(s) exposed statewide	Essentially the entire state is exposed to ambient levels. The largest source of emissions by far in New Jersey is motor vehicles from the traffic that is ubiquitous in the state. Persons living in urban areas, and those living or working near traffic arteries, are likely exposed to higher concentrations than persons in rural areas. The National Air Toxics Assessment (NATA) estimates for 1996[14] show several urban counties in NJ with average concentrations above 0.088 $\mu g/M^3$, while several rural counties have estimated average concentrations below 0.031 $\mu g/M^3$. Interpretation of a map presented in a United Kingdom study suggests that 1,3-butadiene concentrations within 10 meters of heavily traveled roads are in the range of 10 times higher than nearby background concentrations. [15]
Quantification of exposure levels statewide, including populations at significantly increased exposure **(include indoor air as separate category as appropriate)**	An average ambient concentration in the range of 0.07 ppb (about 0.15 $\mu g/M^3$) was measured in Camden in 1997. Concentrations are likely to be lower in less urban regions. An average statewide concentration of 0.07 $\mu g/M^3$, with a range from the 25th percentile to the 75th percentile of about 0.04 to 0.11 $\mu g/M^3$, was modeled by NATA for 1996.[16] Assuming that the average ambient concentration is in the range of 0.07 $\mu g/M^3$ (0.03 ppb) and that a person inhales 20 M^3 air per day, a typical exposure would be about 1.5 µg per day. Current data do not permit characterization of the higher exposures that probably result to persons living or working near heavily traveled roads.
specific population(s) at increased risk	It can be expected that persons whose health is otherwise compromised would be at increased risk. Persons operating motorized equipment, especially 2-cycle engines such as chainsaws, leaf blowers, etc., which appear to have emissions of 1,3-butadiene at least an order of magnitude higher than 4-cycle engines[17], could be exposed to higher levels. There are no manufacturers of 1,3-butadiene in New Jersey; the only industrial releases are modest emissions from refineries. (Of facilities reporting on the 1997 Toxics Release Inventory, the four major NJ refineries reported releasing a total of about 7000 pounds of the chemical.[18]) Therefore, there does not appear to be a population exposed at the workplace.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) **(include indoor air as separate category as appropriate)**	There is insufficient data on variations of ambient concentrations throughout the state. It is likely, based on the range of concentrations measured in urban and rural areas in Vermont that urban concentrations are in the range of 10X higher than rural concentrations.[19]
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	According to the IRIS database[20], the cancer risk level from inhalation, using the linearized multistage procedure, is 1 in 10,000 from a concentration of 0.4 $\mu g/M^3$, 1 in 100,000 from a concentration of 0.04 $\mu g/M^3$, and 1 in

	1,000,000 from a concentration of 0.004 μg/M^3.
Risk Characterization	
risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	Assuming that the statewide average concentration is similar to 0.07 μg/M^3, as modeled by NATA[21], the risk of cancer, based on the IRIS algorithm, is about 1 additional cancer in 48,000 people.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Presumed maximum for these criteria.
size of population(s) affected	State population of about 8,000,000.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	There are the usual uncertainties in extrapolating from high doses in animals to the human population, with the assumption of no threshold. Also, epidemiologic studies for 1,3-butadiene are reported to have yielded conflicting information, with some retrospective cohort studies suggesting an increased incidence of certain cancers and others finding no cases of cancer attributable to exposure to 1,3-butadiene.[22] The exposure estimate includes uncertainty as well. With its relatively short atmospheric lifetime, and association with motor vehicle emission sources, it is likely that concentrations of 1,3-butadiene are much lower in regions of the state less urban than the Camden site at which concentration values have been measured.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	It is probable that further research will clarify the health effects of this chemical. No data was found that implied such potential for this chemical was different from any other chemical. Additional data on ambient or indoor concentrations are likely to be lower than the 0.07 μg/M^3 (0.03 ppb) concentration used for the exposure assessment, and so are expected to lower the risk estimate. However, data better characterizing the atmospheric concentrations near roadways could result in higher risk estimates for exposed sub-populations.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , ≂, where + is improvement)	++ Ambient levels of 1,3-butadiene appear to be considerably lower than formerly. Levels up to 9 ppb were found in Southern California, including Los Angeles, in the mid-60s, and the U.S. average concentration was reported to be 1.5 ppb (about 3.3 μg/M^3) in the late 70s.[23] Concentrations in Vermont in the '93-'95 period[24] were reported to be in the range of 0.1 to 1 μg/M^3 and concentrations in Camden, NJ were measured at 0.224 μg/M^3 (about 0.1

	ppb) in 1990 and at 0.156 μg/M^3 (about 0.07 ppb) in 1997. It appears that there has been an approximately 10-fold decrease in ambient concentrations over the last 20 or 30 years. Recent estimates suggest a continued decline. From 1990 to 1996, as reflected in the Cumulative Exposure Project[25] and NATA,[26] respectively, estimated New Jersey statewide average concentrations decreased from 0.19 μg/M^3 to 0.07 μg/M^3. Levels are also expected to decline to levels 33% to 25% of the 1996 level by 2005 in the U.K.[27] Such a drop in 1,3-butadiene concentrations would be consistent with an observed decrease in carbon monoxide concentrations over the last decade. Statewide levels of carbon monoxide have declined from about 6 ppm in 1990 to about 3 ppm today.[28] Like carbon monoxide, 1,3-butadiene is a product of incomplete combustion, and its concentration in motor vehicle exhaust is reduced to a significant degree by catalytic converters.[29] Additional measures to reduce VOC emissions from motor vehicles due to the need to make further progress in ozone control should further reduce emissions of 1,3-butadiene. National industrial air emissions of the chemical as reported in the toxics release inventory have also declined from about 7 million pounds in 1988 to less than 3 million pounds in 1996.[30] It is also likely that industrial exposures will decline, because the OSHA permissible exposure limit was reduced from 1000 ppm to 1 ppm in 1996.[31]
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	Low. There are no intentional producers of the chemical in New Jersey, so that large concentrations of the material that could result in spills may not exist.
extent to which risks are currently reduced through in-place regulations and controls	Existing controls on VOCs from motor vehicle emissions, including catalytic converters, may be responsible for what appears to be a decreasing level of ambient contamination by 1,3-butadiene. Increased efforts to extend such controls to off-road machinery because of ozone exceedances should result in further reductions in 1,3-butadiene emissions.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
large business/industry	L
small business industry	L
Transportation	H
Residential	L

Agriculture	M (off road motorized equipment)
Recreation	M (personal exposure to 2-cycle engine exhaust from motorboats, etc. could be significant)
Resource extraction	L
Government	L
natural sources	L
Contaminated sites	L
diffuse and non-NJ sources	
Sediment	L
Soil	L
non-local air sources (including deposition)	L
biota sinks	L

Human Health Issue Summary: 1,3-Butadiene

What is it?
1,3-butadiene is a volatile chemical with a gasoline-like odor. It is used in the production of rubber and plastics, and is also a byproduct of incomplete combustion. Motor vehicle exhaust is the largest source of butadiene in New Jersey. Due to its volatility, the impacts of butadiene primarily result from the inhalation of contaminated air (see also the summary for volatile organic compounds). Breathing high levels can cause a range of adverse health effects including blurred vision, nausea, and unconsciousness. Lower concentrations may irritate the eyes, nose, and throat. Butadiene is classified as a known human carcinogen.

What's at risk?
The entire state is exposed to ambient levels of 1,3-butadiene as a result of motor vehicle traffic. People whose health is otherwise compromised will be at greater risk for health effects. Individuals living or working near traffic arteries are likely to be exposed to higher concentrations than rural residents. Individuals operating lawn mowers, motor boats, chainsaws, and other types of motorized equipment could also be exposed to higher levels, as 2-cycle engines appear to emit much greater quantities of butadiene.

What is are the human health impacts in New Jersey?
The average concentration of 1,3-butadiene in outdoor air was measured in Camden in 1997. This concentration (0.07 parts per billion), if extrapolated to the entire state, could be expected to result in 1-2 additional cancers per year statewide. However, concentrations are likely to be lower in less urban areas, and most people spend much of their day indoors, where concentrations are lower. Therefore, measured concentrations are likely to overstate the actual cancer risk.

Issue: 1,3-Butadine
Author: Mike Aucott
Version: 05/21/01

What's being done?

Concentrations of 1,3-butadiene in outdoor air have been decreasing. Like carbon monoxide, butadiene is a product of incomplete combustion, and its presence in automobile exhaust is controlled to a significant degree by catalytic converters. Regulations aimed at reducing ozone levels through the control of VOCs continue to reduce emissions of butadiene.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	**Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)**	**Are there discreter communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)**	**Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)**
M	M	Y	M
3	4	5	3
			3 M

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
stressor	**Acrolein**
description of stressor (including etiology)	Acrolein is an aldehyde by-product of combustion. It is a particularly reactive aldehyde because it contains an unsaturated carbon chain in addition to the reactive carbonyl structure. Acrolein is often associated with other aldehydes produced during combustion, such as from cigarette smoke. Therefore, there are many parallels between an assessment of the effects of acrolein and formaldehyde. (Doull et al.) Many of the effects of formaldehyde are seen when laboratory animals are exposed to acrolein. However, acrolein is more potent in most responses, often by a factor of three (Leikauf).
stressor-specific impacts considered including key impacts	Acrolein is an irritant effecting sensitive mucous membranes. One of the easily recognized symptoms of acrolein exposure is irritation of the eyes (Doull et al.). Acrolein affects lung function and raises the susceptibility towards infection (Leikauf). Lung function changes can be measured by observing expiratory volumes and respiratory resistance (Kutzman). There is some evidence for changes in bronchial cell morphology although the implications of those changes are uncertain (Leikauf).
Exposure Assessment	
Exposure routes and pathways considered **(include indoor air as appropriate)**	The irritant and lung function effects of acrolein are the result of inhalation. However, ingestion is a possible exposure route, although effects from ingestion are largely unknown at concentrations available in the environment. This report does not cover indoor exposures to acrolein, because of a lack of information on indoor exposures.
population(s)/ecosystem(s) exposed statewide	Acrolein is a pervasive pollutant, therefore all New Jersey citizens are exposed. There are higher concentrations of acrolein assumed for urban areas as noted in the National Air Toxics Assessment (NATA) modeling results for the year 1996. (NATA)
quantification of exposure levels statewide, including populations at significantly increased exposure **(include indoor air as separate category as appropriate)**	NATA modeling of acrolein concentrations provides a range of possible exposures for New Jersey counties. The highest reported concentration using this model is in Hudson County showing a median value of .38 ug/m3 (or .17 ppb). Less urban counties such as Sussex and Atlantic show median values of less than .05 ug/m3 (or .02 ppb). Actual monitoring carried out in Camden offers supporting evidence for ambient concentrations possible in urban areas. In 1996, approximately 40 readings showed 13 detections for acrolein at concentrations ranging from .02 to .2 ppb. (The NATA model suggests a median concentration of .07 ppb.) (NJ DEP).

specific population(s) at increased risk	The authors considered the possibility of characterizing effects on those certain populations more susceptible to the irritant nature of pollutants (e.g., those with emphysema, and asthma). In particular, populations sensitive to formaldehyde exposures are likely to be sensitive to acrolein. The characterization of sensitive populations is described under the topic of multiple chemical sensitivities. This controversial health issue is based upon observations of specific populations that suffer physiological responses from concentrations of pollutants that show no impacts on the general population in clinical studies. One characteristic of exposures that is common in those suffering from Multiple Chemical Sensitivities is the presence of chemicals that surpass an odor threshold (Kreutzer et al.). However, most studies of MCS are based on indoor exposures. There was no information available on the responses of sensitive individuals to outdoor levels of irritating chemicals. Children are susceptible to the potential for increased incidence of infection. In New Jersey, the population of children less than age 3 (0-2) is about 330,000 (US Census).
quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations)	There is no differentiation of exposure to children.
Dose/Impact-Response Assessment	
quantitative dose/impact-assessment employed for each population considered	Respiratory rates are decreased in laboratory animals at 1 ppm. Bronchial reactivity as measured by pulmonary resistance is observed in guinea pigs at concentrations greater than 0.8 ppm (Leikauf). Epidemiological studies to identify impacts at lower concentrations that may exist for large populations are notoriously difficult because of confounding factors. Acrolein is a combustion by-product with greatest concentrations in urban areas. These urban areas are also the host to elevated concentrations for dozens of other combustion by-products and other VOCs. There is evidence that increased difficulties with respiratory illness occur in urban areas, much as you would expect with acrolein exposures, but tagging the increase in respiratory illness to a single chemical is difficult. US EPA has determined a reference concentration (RfC) for acrolein equal to .02 ug/m3 (.009 ppb). This estimate is based on animal studies that found possible impacts at 400 ppb under laboratory conditions that are equivalent to 9 ppb human exposures. The impacts from these animal studies include changes in the structure of cells making up the airways. As is the case with many toxicological determinations for regulatory purposes, a factor of 1,000 was included to determine the RfC. (US EPA, (IRIS))
Risk Characterization risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	Using the RfC, populations in Hudson County are exposed to almost 20 times the reference dose for acrolein. Even populations in counties with lowest modeled exposures exceed the RfC. Translating exceedances of RfC determinations is a controversial task, but one way to express an exceedance is that it is not possible to discount the chance that some changes in human health parameters will change based on the exposure. Therefore, in urban counties such as Hudson, there may be hundreds or even thousands of people that will feel the irritant effects of acrolein. For the rest of the state, there is a chance that acrolein contributes to their symptoms of respiratory irritation. In both the urban and less urban cases, there is a chance that no impact results from acrolein exposure.
risk estimate(s) by population at risk	There are no studies estimating proportions of children that may be susceptible to the increased risk of infection. The studies implying the connection simply note that there may be statistical increases in infections among those children exposed to acrolein (Leikauf).

(Children)	
risk estimate(s) by population at risk, cont.	
risk estimate(s) by population at risk, cont.	
assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	L/M - Acrolein exposure is a long term event. (i.e., low levels of the compound are present repeatedly (NJ DEP). The damage that may result accumulates over time, and the ability to repair changes in airway tissue is difficult because of the difficulty in removing exposed individuals from continuing exposures (Lippmann).
size of population(s) affected	Entire New Jersey population (although only a small percentage may feel the effects based on the lack of significant estimates of population effects at reported concentrations). The effects on childhood immune system can be considered for children between the ages of 0-2 (330,000).
assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	M/H - The use of a 1,000 fold uncertainty factor in determining the Reference Concentration and the inability to use epidemiological studies makes the determination of risk from acrolein exposure susceptible to wide ranges of uncertainty. This uncertainty is similar to the determination of risk from several environmental pollutants but in contrast to chemicals with more certain results from epidemiological and laboratory studies such as lead and the other criteria air pollutants.
potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	L/M – There are no greater number of studies focusing on acrolein than other environmental pollutants so it is difficult to speculate that future findings will result in a significant change in the risk estimate. In addition, the co-variance of exposure to other environmental chemicals will make the determination of acrolein-specific damages difficult. As an indoor pollutant, it is possible that acrolein levels may be greater than outdoor levels. If this is the case, then risks due to indoor exposures would be appreciable.
potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =, where + is improvement)	0 - We do not know of any immediate strategy to reduce acrolein emissions other than general strategies to control combustion processes. Therefore, acrolein emissions are not expected to show dramatic changes in the near future. Historical review of acrolein emissions and ambient concentrations have not shown large trends. (NJ DEP)
potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L - We do not believe that acrolein releases from accidental events will generate significantly elevated risks.
extent to which risks are currently reduced through in-place regulations and controls	Unknown.
Relative Contributions of Sources to Risk (H,M,L)	The Cumulative Exposure Project estimates that 50% of acrolein releases come from nonroad mobile sources and an additional 40% are derived from onroad mobile sources. Area sources contribute most of the remainder. Major point sources contribute less than 1%. (US EPA, (CEP))

Allocation of stressor-specific risk to primary NJ sources	
large business/industry	L
small business industry	L
transportation	M/H
residential	L (? Indoor)
agriculture	L
recreation	L
resource extraction	L
government	L
natural sources	L
contaminated sites	L
diffuse and non-NJ sources	Acrolein is a reactive chemical that probably does not travel long distances.
sediment	L
soil	L
non-local air sources (including deposition)	L/M
biota sinks	L

Issue: Acrolein
Author: Ken Jones, Michele Witten
Version: 04/20/01

What is it?

Acrolein is a reactive chemical with a piercing odor that is a by-product of combustion. It is chemically similar to formaldehyde, and has similar effects. It is used in the synthesis of some chemical products, including tear gas, however most of the acrolein in the environment is the result of industrial and vehicle emissions. Acrolein is an irritant, affecting mucous membranes and the eyes. It may also impact respiratory function, particularly in children.

What's at risk?

Acrolein is a pervasive pollutant with higher concentrations in urban areas. Therefore urban areas are at increased risk compared with less urbanized areas in the state. (There are insufficient data on indoor exposures.) Children are more susceptible to a potential increase in infections after exposure and individuals reporting Multiple Chemical Sensitivities (MCS) may be particularly affected by acrolein because of its odor, and the relationship between odor and MCS symptoms.

What are the human health impacts in New Jersey?

Monitoring has shown that exposures in urban areas can be twenty times the reference concentration established by EPA. While these levels are still below the concentration at which laboratory (animal) studies have produced observable health effects, there may be thousands of people that will experience the irritant effects of acrolein. For the rest of the state, there is a chance that acrolein contributes to their symptoms of respiratory irritation. There are approximately 330,000 children under age 3 in New Jersey that are potentially at risk for immune system effects—a subset of these reside in urban areas.

What's being done?

Acrolein concentrations are reduced as the result of general pollution controls on combustion sources.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	**Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)**	**Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)**	**Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)**
Children 2-L/M	2-L/M	2-L/M	2-L/M
Multiple Chemical Sensitivities 3-M	3-M	3-M	1-L
			2-L/M

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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	**Airborne Pathogens**
Stressor	The potential release of biotechnology-related biological materials (*e.g.*, genetically engineered microbes) is also not covered in this report.
Description of stressor (including etiology)	Outdoors: Elevated concentrations of airborne spores of certain types of fungi and actinomycetes [fungi-like bacteria], which are released to the air during wastewater treatment, sanitary landfill operations, composting (of sewage sludge, leaves, or other cellulose-based materials), or various farming practices (Hurst *et al.*, 1997; Atherholt, 1992). High concentrations of bacterial endotoxins are found in organic dusts often generated during various agricultural activities involving the agitation of stored grain, hay, straw, wood chips, tobacco and cotton and during various farming activities including dairy, poultry, hog, and mushroom operations. Compost and animal feed operations can also generate high levels of endotoxins (Olenchock, 1997). Indoors: High concentrations of airborne bacterial endotoxins and fungal spores, mycotoxins and VOCs (Yang and Johanning, 1997). Excessive growth of normally free-living molds, including species of *Penicillium* spp., *Aspergillus* spp*., Paecilomyces, Fusarium* and probably *Stachybotrys atra (*a/k/a *chartarum)*, in presence of warm temperatures and water-saturated (*e.g.*, due to flooding, leaking roofs or plumbing fixtures) or high moisture-impacted (70 % humidity) organic materials such as lumber, dry wall, ceiling tiles, furniture, carpet backing, wallpaper, books, papers, or cellulose-based insulation material, stored hay or leaves such as in barns; and other areas with potentially high mold levels such as antique shops, greenhouses, saunas, mills, construction areas, flower shops, summer homes (CDCa) and in contaminated latex paint (Reuters, 2000). These fungi may also grow in standing/stagnant water in air heating/cooling ducts and/or filters or in humidifiers or in malfunctioning clothes dryer vents using air-derived dust or dirt or clothes lint as a growth medium.

Stressor-specific impacts considered including key impacts	Endemic fungal (including yeast) pathogens (and the disease they cause) include *Histoplasma capsulatum* (histoplasmosis), *Cryptococcus neoformans* (cryptococcosis), *Coccidioides immitis*, (coccidioidomycosis), *Blastomyces dermatitidis*, (blastomycosis),, *Aspergillus* spp. (aspergillosis), *Mucorales, and Zygomycetes* (Yang and Johanning, 1997). Endemic fungi have certain geographical distributions in the US based on climate, soil type, and perhaps other factors (Sternberg, 1994). Most of these pathogens have low levels of incidence in NJ especially for cases of blastomycosis (CDCb). These fungi can cause disease in persons with "normal" immune system function if airborne levels of spores are sufficiently high, but case rates in this population are extremely low. These pathogens more often cause infections in high risk patients (see below). Adverse health effects may include rashes, headaches, fever, chest pain, dry cough, influenza- or, most often, pneumonia-like symptoms or, for cryptococcosis, meningitis. Fatality rates for high risk groups such as AIDS patients are 5 to 12% depending on the disease, except for coccidioidomycosis which has a fatality rate of over 50%. Opportunistic infectious pathogens are not pathogenic to persons with normal immune systems but to immunosuppressed individuals. Such persons include persons on steroid, antibiotic or drug therapies, cancer, organ transplant or AIDS patients, and burn patients. Such individuals are at increased risk of illness from various species of *Aspergillus*, *Penecillium*, *Fusarium,* and *Paecilomyces*, (Ampel, 1996; CDCa; Yang and Johanning, 1997). Symptoms can include fever, shortness of breath, seizures, lung infections (*e.g.*, aspergillosis), lung damage, memory loss, and neurological damage. There is also preliminary (CWRU, 2000; Rao *et al.*, 2000; Vesper *et al.*, 2000), but currently insufficient (MMWRa, 2000) evidence that infants less than 6 months of age are at increased risk for pulmonary hemorrhage (a/k/a hemosiderosis) with chronic cough, congestion, anemia, occasionally resulting in death following exposure to high levels of spores and/or toxins of *Stachybotrys chartarum* (a/k/a *S. atra*).
Stressor-specific impacts considered including key impacts (con't)	Fungi produce over 400 known mycotoxins of the alkaloid, cyclopeptide and coumarin chemical groups. Such toxins include trichothecenes, aflatoxins, ochratoxins, zearalenone, patulin, and citranine among others (Tuomi *et al.*, 2000). Toxin producing fungi include species of *Penicillium, Aspergillus, Fusarium, Tricothecium,* and *Stachybotrys chartarum* among others. Mycotoxin-containing fungi have been found in more than 40% of water-damaged building materials in a Finnish study (Toumi *et al.*, 2000). Symptoms of toxin exposure are neither allergic nor infectious in nature and can be acute or chronic. Acute effects can include dermatitis, cold or flu-like symptoms, sore throat, headaches, fatigue, diarrhea and impaired immune system function which can predispose an individual to opportunistic infections. In animal studies, some mycotoxins have been shown to possess serious chronic affects including organ and tissue damage, teratogenicity (zearalenone), and mutagenicity (aflatoxins)(Yang and Johanning, 1997).
	Certain fungal spores are also strong allergens. Such diseases include asthma, hay fever, allergic contact dermatitis, and pneumonitis. Allergenic agents include species of *Aspergillus, Alternaria, Botrytis, Cryptostroma, Penicillium, Mucor, Trichosporon, Paecilomyces* and others (Yang and Johanning, 1997). Symptoms can include eye, nose and throat irritation, nasal stuffiness, sinusitis, wheezing. More serious symptoms include fever and shortness of breath,

	allergic contact dermatitis (CDCa). Finally, bacterial endotoxins, which are lipopolysaccharide complexes from the outer membranes of certain bacteria are stable, ubiquitous environmental compounds that, when present in sufficiently high concentrations, can trigger a neutrophil and/or pulmonary macrophage immune response resulting in acute (including mucous hypersecretion and reduced lung function) and chronic (bronchitis) effects (Olenchock, 1997).
Exposure Assessment	
Exposure routes and pathways considered **(include indoor air as appropriate)**	Inhalation (including indoor air)
Population(s)/ecosystem(s) exposed statewide	Exposure to low levels of airborne fungal spores is universal, but disease is uncommon. Disease is the result of challenge dose, duration of exposure and health status of the exposed individual (see below). The total population of sensitive individuals in NJ is not known. For example, the CDC offers no national estimate of community-acquired or nosocomial aspergillosis (CDCc). Because fungi are relatively new as a threat to human health (due to emergence of AIDS, increases in organ and bone marrow transplants and the like), none of the 49 state health departments that report disease surveillance data to the CDC report any fungal diseases. CDC maintains a National Nosocomial Infections Surveillance System (NNISS) which collects data on hospital-acquired infections from a network of 285 "sentinel" hospitals, but data specific to fungal infections is not available in their published reports (MMWRb, 2000; CDCe).
Quantification of exposure levels statewide, including populations at significantly increased exposure **(include indoor air as separate category as appropriate)**	Concentrations causing illness are significantly higher than outdoor levels. Background levels of mold and actinomycete spores and endotoxins are low. Concentrations of <u>all types</u> of airborne fungal spores are usually in the range of 10^3-10^4 colony forming units per cubic meter of air (CFU/m^3) (Lacey and Crook, 1988). Background levels of *Aspergillus fumigatus* (Af) spores have been observed in the range of 0 to 120 CFU/m^3, with concentrations generally < 20 CFU/m^3. Thermophilic fungi; range, 0 - 193 and median, 2-3 CFU/m^3. Mesophilic fungi, range, 0-7220 and median, 324 - 325 CFU/m^3 (studies reviewed in Atherholt, 1992). Transient concentrations of fungal spores and bacterial endotoxins can be quite high for some persons working at or living near open-air composting facilities or involved in certain other occupations:

Specific population(s) at increased risk	Concentrations of *Aspergillus fumigatus* ("Af") at or near open-air compost facilities can be high during operational periods (10^3 - 10^6 CFU/m^3), but low during non-operational periods (Atherholt, 1992). One modeling study showed that during "non-dispersive" atmospheric conditions, Af concentrations could be one order of magnitude higher than background levels up to 1 kilometer from a compost source. Under "dispersive" conditions, background levels would be achieved within 0.6 kilometers from the site. Data from other studies show compost Af levels reach background levels at a distance of roughly 150 meters (Atherholt, 1992). Allergy threshold levels to common molds have been reported to be between 100 CFU/m^3 for *Alternaria* spp. and 3,000 CFU/m^3 for *Cladosporium* spp. Spore concentrations in the range of 2,000 to 20,000 CFU/m^3 for *Aspergillus, Penicillium* and *Paecilomyces* were observed in a compost worker with alveolitis and fibrosis (Yang and Johanning, 1997). Bacterial endotoxin threshold concentrations for reduced lung function in one study were calculated to be in the range of 90 - 330 endotoxin units (EU) / m^3 (9-33 ng/m^3). In comparison, various dust-inducing agricultural activities can result in airborne endotoxin levels ranging from 4,500 - 88,500 EU/m^3. Chronic effect thresholds were observed to be in the range of 10-200 EU/m^3 (1 - 20 ng/m^3) in a second study and 0.2 to 470 ng/m^3 in a third study (Olenchock, 1997). Two endemic fungal pathogens, *Histoplasma* and *Cryptococcus* (and the bacterial pathogen *Chlamydia psittaci*) are commonly associated with aged bird droppings (or soils contaminated with same) or fresh or aged bat droppings (CDCc). For these pathogens, at-risk groups include infants, young children, and elderly, particularly those with chronic lung disease or persons with cancer or AIDS. A variety of occupational groups have a higher than normal chance of exposure to high concentrations of bird or bat droppings (*e.g.*, bridge inspector, pest control worker, cave explorer, poultry farmers, etc.). Such persons should wear respirators which protect against the inhalation of disturbed, aerosolized bird or bat droppings (CDCd). Certain occupations expose workers to unsafe levels of fungal spores. These occupations include wastewater treatment plant, sanitary landfill and compost workers, farmers, some greenhouse workers (Yoshida *et al.*, 1993). Persons living near wastewater treatment plants, sanitary landfills composting facilities and farms may also be at risk. Other populations at significantly increased exposure to *Aspergillus* include some farmers, antique shop workers, greenhouse/flower shop workers, and anyone occupying a structure with excessive mold spore levels as described above. Owners of some summer homes in high moisture areas, such as the coast, may be subject to higher exposure levels. For opportunistic infectious pathogens, at risk groups include immunosuppressed individuals. Such persons include bone marrow and organ transplant patients and patients with leukemia or lymphoma receiving radiation and chemotherapy. Persons receiving antibiotics, steroids and certain other drugs, or with diabetes or with another underlying disease (*e.g.*, liver or kidney disease, AIDS patients, burn victims).
	In addition persons with asthma, allergies, or other immunological disorders often have high sensitivity to fungal

	allergens. Reported percentages of the population with allergy to molds vary from 2 to 18% with approximately 80% of persons with asthma allergic to molds. (Yang and Johanning, 1997).
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) **(include indoor air as separate category as appropriate)**	Statewide population of persons at increased risk, such as those with asthma, allergies, diabetes mellitus, persons with weakened immune systems and possibly infants less than 6 months old, that are exposed to unsafe levels of mold spores in indoor air, is not known.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	Unknown. Airborne spore concentrations that cause no effect in some people may cause mild or severe adverse effects in other people (*e.g.*, persons with asthma). Interindividual differences in susceptibility are common and can be quite large. The dose-response of humans to airborne mycotoxins is not known (Toumi *et al.*, 2000).
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	not known
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Frequency of severe symptoms (*e.g.*, pulmonary hemorrhage, neurological effects, death) is probably low. Frequency for milder symptoms may also be low, but is likely to be higher than for severe symptomology.
Size of population(s) affected	Unknown
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	H
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	H (currently very little data)
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, -, =, □ where + is improvement)	0
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L
Extent to which risks are currently reduced through in-	The NJDEP has no regulations controlling levels of indoor air pathogens (J. Held, personal communication).

place regulations and controls	The federal sludge "503" regulations which concern the use and disposal of wastewater treatment biosolids do not address airborne pathogens such as those that may occur at wastewater treatment or sludge composting facilities (USEPA, 1993). Personal respiratory protective equipment guidance and indoor air quality information is offered by the National Institute for Occupational Safety and Health, the Association of Occupational and Environmental Clinics and the American Industrial Hygiene Association, the USEPA Indoor Environments Division, local or county health departments and the NJ Department of Health and Senior Services.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	Low at most locations, but possibly high at a few locations (*e.g.*, water-damaged buildings, contaminated heating/cooling airflow systems) and in certain industries (*e.g.*, composting facilities).
Small business industry	Low at most locations, possibly high at a few locations (*e.g.*, water-damaged buildings, contaminated heating/cooling airflow systems).
Transportation	Low in most carriers, but possibly high in a few with contaminated air handling systems.
Residential	Low in most homes but high in homes with water damage or heating/cooling system problems.
Agriculture	Medium. Autocomposting of manure and the like. High levels in areas with stored hay and grains (*e.g.*, barns).
Recreation	none
Resource extraction	none
Government	Low at most locations, possibly high at a few locations (*e.g.*, water-damaged buildings, contaminated heating/cooling airflow systems).
Natural sources	High. All airborne fungi derived from natural sources.
Contaminated sites	none
Diffuse and non-NJ sources	
Sediment	none
Soil	Low. The source of *Coccidioides immitis*
Non-local air sources (including deposition)	none. No long-range transport to any measurable extent.

Biota sinks	Medium. Decaying vegetation.

Human Health Issue Summary: Airborne Pathogens

What are they?

Airborne pathogens include fungal spores and bacteria that are released to the air during wastewater treatment, sanitary landfill operations, composting, and various farming practices. Sources of these pathogens include fungal growth associated with warm wet areas, and agricultural activities that generate large quantities of organic dusts that may contain high concentrations of bacterial toxins. Some pathogens are associated with bird or bat droppings. Human health effects include respiratory infections, allergic responses, eye, nose and throat irritation, as well as more severe cases involving fever and shortness of breath. Related reports include, Legionella, Hantavirus, indoor microbial pollution, and indoor asthma inducers.

What's at risk?
Exposure to low levels of airborne spores is universal, but disease is uncommon. Infants and the elderly are particularly subject to fungal infections. Individuals with compromised immune systems or other underlying disease are more susceptible to the health effects that may result from exposure to airborne pathogens. Asthmatics are also especially sensitive to fungal allergens. Workers near concentrated sources such as composting facilities are at increased risk of exposure, as are people who are occupationally exposed to higher than normal concentrations of bird or bat droppings.

What are the human health impacts in New Jersey?
There is very little information regarding the number of illnesses that may be attributed to airborne pathogens. Thresholds for allergic response are regularly exceeded near composting facilities. In some cases, elevated levels may exist up to 1 kilometer from the facility. Dust-inducing agricultural practices can produce concentrations of bacterial toxins far in excess of those known to impact lung function. Natural sources such as bird and bat droppings may also result in localized exposures to elevated levels of pathogens.

What's being done?
There are no regulations on airborne pathogen generation. Occupational guidance is available for protecting workers from exposure.

Severity of specified health effects at current levels of exposure (H,M,L)	**Size of population at significant risk for each health effect (H,M,L)**	**Are there discrete communities at elevated risk? (Y,N)**	**Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L)**
Low. 1.	Unknown.	Yes.	Low to medium? 2
			Low to medium? 2

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CDCb. Centers for Disease Control and Prevention's public health images website: "http://phil.cdc.gov/public/". Blastomycosis = image 491; Histoplasmin = image 452; Coccidiodin = image 474.

CDCc. Centers for Disease Control and Prevention's disease information website: "http://www.cdc.gov/ncidod/dbmd/diseaseinfo."

CDCd. Centers for Disease Control and Prevention's National Institute for Occupational Safety and Health website: "http://www.cdc.gov/niosh."

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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Hazard Identification	
Stressor	**Arsenic**
description of stressor (including etiology)	Arsenic is a metalloid. It belongs to the same element group as Phosphorus. Arsenic compounds occur in many rocks and thus find its way to the soil, water, and food, being especially high (3 to 170 ppm) in some seafood. Arsenic is a normal constituent of the human body. However we lack knowledge of biochemical mechanism and physiological role of arsenic. Arsenic is a trace element (believed to be essential for life) and is found in tissues in minute amounts. Trace element deficiencies are rare, because any diet that is adequate in other respects easily supplies the needed minerals. The use of arsenical insecticides in agriculture has decreased greatly.(1)
stressor-specific impacts considered including key impacts	Experimental exposure: The acute effects of arsenic in animals are similar to those observed in humans. The degree of irritation of the gastrointestinal tract involved in poisoning by arsenic trioxide depends on its purity. Dogs fed sodium arsenate at a rate of about 2.7 mg/kg/day showed anorexia, listlessness, and weight loss leading to cachexia, and eventually death. Neither skin lesions nor polyneuropathy has been observed in experimental animals. Thus except for weight loss and anemia in some species, arsenic poisoning effects as it occurs humans is dissimilar to that in animals. Human exposure: This is normally due to therapeutic use (in the past) or accidental and intentional poisoning (for many years arsenic has been the most important single cause of accidental deaths associated with pesticides in the U.S.). Death may result from severe hypotension and collapse as in “dry” cholera. If the patient survives the acute phase, skin eruptions, a moderate depression of blood cells, and polyneuropathy (paresthesia, pain, burning, and tenderness of the affected limbs leading to loss of proprioception) may appear. Dermatitis is especially prominent in the palms and soles and other subject to pressure. White transverse bands in the nails (Mees lines) frequently appear in about 6 weeks and may accompany polyneuropathy, which often appear 1-3 weeks after ingestion. When Mees lines occur in conjunction with polyneuropathy, it is almost pathognomonic of arsenic poisoning. Ingestion of inorganic arsenic is an established cause of cancer in humans, based on epidemiological data from Taiwan, Bangladesh, Chile, and Argentina. Two histopathological types of skin cancer have been associated with arsenic-squamous carcinoma in keratin areas and basal cell carcinoma. Skin cancers caused by arsenic differ from those resulting from ultraviolet light by occurring in areas of the body not exposed to sunlight, e.g., soles of the feet.

	Hyperpigmentation is followed by hyperkeratosis. The latent period for the initiation of exposure to appearance of skin cancer ranges from 6 to 50 years when arsenic was used medicinally, e.g., Fowler's solution. When the exposure was from contaminated drinking water, the shortest latency period was about 24 hours. (1) Both the EPA and the National Toxicology Program (NTP) have classified arsenic as a known human carcinogen. (7) Arsenic has also been associated in nephrotoxicity and lung cancers. (5) & (11) More recent evidence has shown that arsenic can also cause cancers of the liver, kidney, and urinary bladder in humans (NRC*)*.
Exposure Assessment	
exposure routes and pathways considered (include indoor air as appropriate)	Inhalation: Pulmonary system due to emissions from manufacturers. Arsenic has been implicated in lung cancer due to occupational exposure in arsenic pesticides manufacturing and copper smelting factories. Inhalation exposure to arsenic trioxide has been found to increase the frequency of chromosomal aberrations in peripheral lymphocytes of smelter workers.(7) Oral: Naturally occurring arsenic is found in groundwater in many parts of the world including Taiwan, Bangladesh, Chile, Argentina, and parts of the United States. Contamination of well water by arsenical pesticides has been cause of outbreaks of poisoning in Russia. (1) The Environmental Defense Organization's ranking of States by their total Pounds of arsenic released (scorecard.org) excludes New Jersey from the list of top 18 highest releasers in the country.
population(s)/ecosystem(s) exposed statewide	NJDEP estimates that up to 5% of State's acreage (240,000 acres) may be impacted by historical use of arsenical pesticides, and inadvertent ingestion of contaminated soil particularly by children is a prime concern. (2, page 8) Inorganic arsenic does not break down and therefore will persist in the environment indefinitely. Arsenical pesticides are not particularly water-soluble and therefore pose minimal threat to deep ground water. However, these pesticides may pose some risk to shallow aquifers in acidic or sandy soils. The NJDEP's analysis of soil sampling data from 18 selected current and former agricultural sites in New Jersey show: Between 1996 and 1998 shows that arsenic was detected in all the samples (463) in the data set at concentrations ranging from 1.4 ppm to 310 ppm. Arsenic was detected above the Department's residential soil action criteria more frequently than any other analyte. Arsenic was detected above the action criterion-(_20 ppm) in 38% of the samples. A Department survey of background concentrations of arsenic in soil in rural and suburban areas of the state shows that, non-agricultural soils contained 0.02-22.7 ppm with an average of 3.25 ppm. (2,pages 8-17).

	Soil samples taken from 18 sites in New Jersey showed that only the township of Upper Freehold in Monmouth County (site#7, 84 acres, orchards and field crops) had any significant arsenic concentrations due to natural background arsenic (range <20-55 ppm, median 35.5 ppm, number of samples: 5, frequency of detection: 5 with 2> criteria). (2, pages 10-11). The number of individuals exposed to elevated arsenic levels in drinking water could be quantified from the data discussed below, but this has not been done as yet.
quantification of exposure levels statewide (include indoor air as separate category as appropriate)	The Bureau of Safe Drinking Water, NJDEP, data on public working systems from the period 1993-1999 shows the following: No samples exceeded the current drinking water standard of 50 µg/L. The current standard is expected to be revised in the near future to a level between 2 µg/L and 10 µg/L. 48 of 357 (13%) of well facilities tested exceeded 10 µg/L., 129 of 357 (36%) of well facilities exceeded 5 µg/L, and 240 of 357 (67%) of well facilities exceeded 2 µg/L. In an ongoing study of private wells in the Piedmont Physiographic Province, very few private wells exceed the current standard of 50 µg/L, but a significant percentage of wells are anticipated to exceed the revised standard when it is proposed. EPA's proposed new regulation will lower by 90% the maximum limit of arsenic for drinking water to 5 ppb from 50 ppb.
specific population(s) at increased risk	Accidental ingestion of contaminated soil by children. (2) Individuals with elevated arsenic levels in their public water supplies or private wells. Most accidents in industry have involved the conversion of inorganic arsenic to arsine gas. Workers in metal smelters and nearby residents may be exposed to elevated inorganic arsenic levels from arsenic released into the air. (5) Based on 1990 Modeled Concentration of the Cumulative Exposure Project (Attachment 1), Hudson County has highest arsenic concentrations in New Jersey. The Mean and Median of the 161 tracts considered were 0.001470 and 0.001398 ug/m^3 respectively, (Min.: 0.000925, P25: 0.001219, P75: 0.001635, Max. 0.002329 ug/m^3). A more recent survey by the EPA (National-Scale Air Toxics Assessment, NATA) generated the 1996 Modeled Median Ambient Concentrations (MAC) for arsenic compounds (attachment 2) (www.epa.gov/ttn/atw/nata). A comparison of the data from 1990 CEP and 1996 NATA provides noteworthy patterns: 14 New Jersey counties improved (reduced) their MAC of arsenic (mainly northern NJ). These were Passaic, Bergen, Essex, Union, Middlesex, Monmouth, Ocean, Cape May, Burlington, Hunterdon, Somerset, Warren, Morris and Sussex.
	Atlantic was the only county that showed a slight increase in MAC.

	Six counties (mainly southern) maintained 1990 median concentrations (within MAC range specified in the 1996 NATA). These were Cumberland, Salem, Gloucester, Camden, Mercer and Hudson counties. Six counties ranked in the 95th Percentile (top 5%)in the nation with 1996 MACs in the range of 0.00018 – 0.018 micrograms per cubic meter. These were Hudson, Atlantic, Cumberland, Salem, Gloucester and Camden. "EPA strongly cautions that these results should not be used to draw conclusions about local concentrations or risk. The results are most meaningful when viewed at the state or national level, for smaller areas, the modeling becomes less certain. In addition, these results represent conditions in 1996 rather than current conditions."
quantification of exposure levels to population(s) at increased risk (due to factors other than exposure) (include indoor air as separate category as appropriate) Dose/Impact-Response Assessment quantitative dose/impact-assessment employed for each population considered	Low. Methylation of arsenic by human body may be regarded as a detoxification process, as the methylated derivative is less toxic than the inorganic form. Factors affecting methylation are therefore important. Populations having certain dietary deficiencies (e.g., methionine) may be more susceptible to arsenic poisoning. (1) Effects of organic arsenic on human health are, however, considerably less pronounced: Inhalation exposure- No studies were located regarding death in humans after such exposure. Additionally, no studies were located regarding cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, dermal, immunological, neurological, reproductive, developmental, genotoxic and cancerous effects secondary to such exposure. (7) Oral exposure- The only human effects manifested by male subjects exposed to a one time exposure of 77.1 mg/kg/day of organic arsenic were sinus tachycardia and gastrointestinal symptoms(vomiting, abdominal pain, hyperactive bowel & watery garlic-smelling stools). No studies were located regarding death in humans after oral exposure to organic arsenicals. (7) A dose of 5-50 mg of arsenic trioxide is toxic. A dose of about 1.8 mg/kg is said to have proved fatal to an unhabituated adult, but recovery has occurred after much larger doses. Very young children may be more susceptible. Ant bait with 0.61% sodium arsenate solution at a dosage of 1 mg/kg can cause serious illness in a child and 2mg/kg can cause death. (1, page550) Estimates of the minimum lethal dose in humans range from 1-3 mg As/kg/day, although there may be considerable variation between individuals. (7, page135) Cancer associated with arsenic has not been reported at air concentrations of 0.1 mg/m^3 or less even for long periods. It has been estimated that exposure to air concentrations of 50μg As/m^3 occupationally for 24 years would result in a 200% excess risk of lung cancer (WHO,1981). (1) The current arsenic oral slope factor in the USEPA IRIS data base of 1.5 (mg/kg/day)$^{-1}$ is based on human skin cancer. If all types of cancers were considered together, this factor would be higher (NRC). Based on the slope factor in IRIS, the lifetime cancer risk from drinking water containing 2 μg/L arsenic is 1 x 10^{-5}, (new EPA limit is 5 μg/L). The inhalation unit risk factor for cancer in IRIS of 4.3 x 10^{-3} (mg/m3)$^{-1}$ is based on human lung cancer. Based on this, the risk of inhaling arsenic for a lifetime at 2 x10^{-2} ug/m3 is 1 x 10
Risk Characterization	

risk estimate(s) by population at risk including probability and number of cases/occurrences (Specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	Based on estimates of routine emissions state wide, the 1990 EPA exposure/cancer estimates for New Jersey: -State maximum cancer risk: 21 in one million of population. -State mean cancer risk: 3 in one million of population. -County average risk: 15 counties have an average risk of 1 in a million or more. Hudson county has the highest, with an average cancer risk from arsenic exposure of 6 in one million of population. Using the EPA estimate of carcinogenic strength, the concentration of arsenic below which the cancer risk is less than one additional case in one million people exposed for a lifetime, corresponds to a lifetime exposure to a soil concentration of 0.4 ppm for residential exposure. The NJDEP's soil action criteria for arsenic(20 ppm) is based on the naturally occurring background levels. Translated into cancer risk based on EPA assumptions and calculations, a person exposed to 20 ppm of arsenic has a 50 in one million chance of getting cancer over a lifetime due to arsenic exposure alone.(2) The information given above on cancer slope factor could be combined with data on number of people exposed to elevated arsenic levels in drinking water to estimate number of expected excess cancers.
assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Arsenic is not broken down or destroyed in the environment. However, it can change from one form to another by the action of bacteria that live in the soil or water. (7) Arsenic is rapidly cleared from human blood and tissue levels change rapidly after a single dose. Inorganic arsenic is very toxic to mammals and has been assigned to Toxicity Class 1 based on oral toxicity tests (Farm Chemicals Handbook, 1989). Inorganic arsenic also has been classified as human carcinogen (A) (IRIS, 1995) and long-term effects include dermal hyperkeratosis, dermal melanosis and carcinoma, hepatomegaly, and peripheral neuropathy(NAS 1991). (12)
size of population(s) affected	Difficult to discern without a wider survey and more sampling data. This however may change with more data becoming available .
assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	Medium There are many uncertainties about the shape of the dose-response curve for cancer from arsenic, and whether it is linear at low doses. The shape of the curve greatly impacts the risk estimates for exposures at low doses.

	Ingestion of shrimp with high natural (organic) arsenic content cause blood levels to rise. Urinary arsenic (inorganic) levels in occupational exposure are usually in the range of hundreds of micrograms per liter, values that can easily be confounded by ingestion of seafood. A better index of exposure to inorganic arsenic may be obtained by measuring the urinary concentrations of inorganic arsenic and the two metabolites mono- and dimethyl arsenic.(1)
potential for future changes in the underlying risk from this stressor, (+++, ++, +, 0, !, =,/ where + is improvement)	+ EPA has adopted new, more stringent drinking water regulation for arsenic (2/22/02). This should result in lower exposure and risk.
potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	Low Rupture of baghouses/collection systems in copper smelting facilities. A study of worst-case release scenario for facilities handling large quantities (and/or near high population density areas) may be advisable. The severity of impact depends on the quantity released and the proximity to population centers. Although the impact potential may be high, the probability of such an accident is nonexistent since there are no longer any copper smelters operating in New Jersey (TRI and ISRA).
extent to which risks are currently reduced through in-place regulations and controls	Medium In light of EPA's new proposed regulation for lowering the acceptable arsenic content in the drinking water this ranking has been upgraded to Medium. The use of arsenical pesticides has been discontinued. Current air & water pollution control regulations. The NJDEP Residential Soil Cleanup Criteria for Arsenic (20 ppm). (2)
Relative Contributions of Sources to Risk (H,M,L)	

Allocation of stressor-specific risk to primary NJ sources	The Facility & Chemical Inventory Tracking System (FACITS), survey year 1997 shows a tabulation of 49 facilities in New Jersey with arsenic / arsenic compounds inventory. Nineteen facilities reported inventory quantities in the range of 101- 1000 pounds or higher. The arsenic itemization threshold quantity is 500 pounds or higher.
large business/industry	M -3 The Toxic Release Inventory (TRI) has shown only U.S. Fuji Electric Inc. of Piscataway, NJ, to have routine releases of arsenic in Fugitive Air in the amount of 5 pounds (1995), 2 pounds (1996) and 2 pounds (1997) with a population exposure of more than 440,000 per release incident. (3) The 1996 New Jersey arsenic emission data shows a total of 7.10 Tons per year of arsenic discharged into air by various industry groups. The coal burning Utility Boilers (i.e. B. L. England) have by far the largest arsenic emissions (6.4 Tons per year). (8)
small business industry	M -3 Arsenic is also used in the battery manufacturing, wood preserving, primary metal and fabricated metal products (electroplating, plating, polishing, anodizing, and coloring) industries. In New Jersey these industries have to comply with certain pretreatment standards & effluent guidelines for their discharge. Their discharge will then be treated at a treatment plant. Therefore arsenic is a low risk in this category. However it should be noted that if biocides are discharged in the effluent they might pose a threat to the digesters in the sewage treatment plants and reduce their efficacy.
Transportation	L -1
Residential	L -1
Agriculture	M -3 Lead arsenate was used at a rate of several pounds per acre in apple and other fruit orchards, vegetable fields, golf courses and turf farms in New Jersey until 1967. (2) Six counties (Burlington, Cumberland, Gloucester, Hunterdon, Monmouth, and Salem) have provided most of the

	fruit production over the last 90 years. (2)
Recreation	L/M -2 Golf courses and turf farms.(2)
resource extraction	L/M -2 Arsenic contamination in water is often a by-product of mining and extraction of metals such as copper, gold, lead, zinc, silver, and nickel. This contamination will continue to grow as high-grade ores with low arsenic content are being depleted and processing of sulphide ores with high arsenic content becomes increasingly common. In most cases, it is not economical to recover arsenic contained in process streams because there is little demand worldwide for arsenic. Arsenic can be present in leachates from piles of coal fly ash, in contaminated groundwaters, in geothermal waters, and in acid drainage from pyritic heaps that result from the past practices of mining of metallic ores. (4)
Government	L -1
natural sources	M/H -4 Much of the arsenic found in drinking water arises from natural geologic formations. One of the inherent problems with the presence of arsenic in soil is that arsenic is a naturally occurring metal and is often difficult to distinguish between concentrations from application of pesticides and those that occur naturally(2)
contaminated sites	M -3 Since 1960, Gloucester and Burlington have been the largest fruit-producing counties, therefore at a higher risk for soil contamination due to past use of arsenical pesticides. (2) Other examples of sites with significant arsenic contamination are: Novartis Pharmaceutical (Union County) from overuse of arsenical pesticides, Rockland Chemical Company (Essex County) which packaged such pesticides and Kearny Smelting Company (Hudson County). Unfortunately at this time DEP does not have contaminant specific database in the SRP except for mercury.
diffuse and non-NJ sources	
Sediment	H -3, in specific locations M -2 statewide. (10)

Soil	Characterization of Ambient Levels of Selected Metals and other Analytes in New Jersey Soil, Table 8, Summary statistics for arsenic in soil within the Piedmont Province, NJ, BEM Systems, Inc., Florham Park, NJ, May 1997. (9) Method Detect. Lt. / Instr. Detect. Lt.= 0.13. Practical Quantitation Lt. = 1.0 Units: mg/kg	Frequency	67/67
		Average	10
		Median	5.2
		Minimum	1.7
		Maximum	49.7
		90% Percentile	24.2
		95% Percentile	29.5
		Geo mean	7.0
		Geo Standard Dev.	2.22
		Lower 95th Percent Confidence Limit (LCL).	5.76
		Upper 95th Percent Confidence Limit (UCL).	8.43
	Characterization of Ambient Levels of Selected Metals and other Analytes in New Jersey Coastal Plain Region Soils, Table 8, Summary Statistics for Metals (arsenic) in Soil within NJ Coastal Plain Province, BEM Systems, Inc., Chatham, NJ, October 1998. Method Detection Limit:1 mg/kg(ppm).	Number of samples	95
		Detects	86
		Mean	7.9
		Median	5.2
		Minimum	0.5
		Maximum	83.1
		90th Percentile	13.6
		95th Percentile	15.7
		Geometric Mean	4.7
		Standard Deviation	11.7
		Lower 95th percent confidence limit	5.5
		Upper 95th percent confidence limit	10.3
non-local air sources (including deposition)		M: 3 Arsenic is globally transported from mining, manufacturing, and fossil fuel burning.	
biota sinks		M -3 Arsenic is cycled through most animals, some animals and plants may take up some forms of arsenic, however the role of Rhizofiltration (the use of plants to remove heavy metals from aqueous streams) and Phytoextraction (the use of plants to remove heavy metals from soils) needs further research.	

	Total arsenic (inclusive of both inorganic and organic forms) has been included in six national monitoring programs; however, no national program is currently monitoring total inorganic arsenic in fish or shellfish tissues. Arsenic and arsenic-containing organic compounds have not been shown to bioaccumulate to any great extent in aquatic organisms. Because it is the concentration of the inorganic arsenic in fish and shellfish that poses the greatest threat to human health, EPA recommends that total inorganic arsenic (not total arsenic) be analyzed in contaminant monitoring programs. (12)

Human Health Issue Summary: Arsenic

What is it?
Arsenic is a trace element normally found in soil, water, food, and the human body. Like other trace elements, minute amounts found in human tissue are believed to be essential for life. The former widespread use of arsenic in pesticides and for copper smelting has resulted in greater concentrations of arsenic in certain areas. An inorganic form of arsenic, arsenic trioxide, is a known human carcinogen and is associated with cancers of the lung, skin, liver, kidney, and bladder. Toxic amounts of inorganic arsenic may also cause neurological disorders.

What's at risk?
Up to 5% of New Jersey's land acreage may be impacted by historical use of arsenical pesticides, and inadvertent ingestion of contaminated soil by children is a prime concern. Lead arsenate was a pesticide used in fruit orchards, vegetable fields, golf courses, and turf farms. Others at risk include individuals with elevated arsenic levels in their public water supplies or private wells, and industrial workers exposed to inorganic arsine gas released into the air.

What are the human health impacts in New Jersey?
A large fraction of the New Jersey population is exposed to slightly elevated levels of arsenic in the air. About 5 million residents are potentially at risk due to ground water sources of drinking water. Estimates show as many as 2 additional cases of cancer per year due to air exposures and 4 per year related to arsenic in drinking water.

What's being done?
The use of arsenical pesticides has been discontinued. Arsenic is included in federal regulations on air emissions, hazardous waste, and other environmental programs. In 2001, EPA reduced the acceptable level of arsenic in drinking water from 50 parts per billion to 10 ppb. New Jersey DEP has adopted a soil cleanup standard to apply in remediation of hazardous sites and has convened a task force to address historic pesticide contamination.

Risk Ranking Table
Arsenic

Severity of specified health effects at current levels of exposure (H,M,L)	Size of population at significant risk for each health effect (H,M,L)	Are there discreter communities at elevated risk? (Y,N)	Overall risk ranking (as a function of severity and population effected integrating across health

(also 1-5 with 1 being least severe)	(also 1-5 with 1 being smallest)	(also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
3 M	3 M	3 Y	3 M
			3 M

This ranking would most likely change as more data becomes available.

Tracts represents the number of census tracts in 1990 for each county.

Mean represents the arithmetic mean of all 1990 Modeled Concentrations for each county and Median the median. Min represents the minimum 1990 Modeled Concentration for each county, P25 the 25th percentile, P75 the 75th percentile, and Max the maximum. All concentrations are in micrograms per cubic meter [gg/m3]. ;.

There are 21 counties in this state.

BBS/u/EPA1
[Cumulative Exposure Project Home *I* EPA Home *1* Search *1* What's new/ The Cumulative Exposure Project - Data Table

Please send comments about this page to axelrad.danielQ.epa.gov

County	*Tracts*	*Mean*	*Median*	*Min.*	*P25*	*P75*	*Max*
Atlantic County	*71*	*0.000122*	*0.000117*	*0.000056*	*0.000092*	*0.000148*	*0.000269*
Bergen County	*210*	*0.000714*	*0.000669*	*0.000287*	*0.000506*	*0.000893*	*0.001513*
Burlington County	*110*	*0.000378*	*0.000370*	*0.000078*	*0.000280*	*0.000460*	*0.000828*
Camden County	*120*	*0.000635*	*0.000602*	*0.000213*	*0.000413*	*0.000843*	*0.001280*
Cape May County	*23*	*0.000077*	*0.000069*	*0.000036*	*0.000062*	*0.000091*	*0.000151*
Cumberland County	*29*	*0.000412*	*0.000277*	*0.000091*	*0.000169*	*0.000640*	*0.001590*
Essex County	*225*	*0.001280*	*0.001036*	*0.000483*	*0.000880*	*0.001298*	*0.004874*
Gloucester County	*60*	*0.000354*	*0.000316*	*0.000160*	*0.000256*	*0.000439*	*0.000695*
Hudson County	*161*	*0.001470*	*0.001398*	*0.000925*	*0.001219*	*0.001635*	*0.002329*
Hunterdon County	*22*	*0.000198*	*0.000165*	*0.000101*	*0.000140*	*0.000199*	*0.000467*
Mercer County	*63*	*0.000657*	*0.000573*	*0.000268*	*0.000460*	*0.000762*	*0.002234*
Middlesex County	*177*	*0.000568*	*0.000514*	*0.000264*	*0.000422*	*0.000647*	*0.001396*
Monmouth County	*147*	*0.000286*	*0.000270*	*0.000105*	*0.000212*	*0.000340*	*0.000670*
Morris County	*89*	*0.000309*	*0.000315*	*0.000111*	*0.000234*	*0.000377*	*0.000134*
Ocean County	*87*	*0.000112*	*0.000106*	*0.000048*	*0.000087*	*0.000647*	*0.000224*

Issue: Arsenic
Author: B.B. Salahi
Version: 06/20/01

Passaic County	*93*	*0.000651*	*0.000663*	*0.000109*	*0.000504*	*0.000778*	*0.002433*
Salem County	*23*	*0.000369*	*0.000335*	*0.000162*	*0.000221*	*0.000495*	*0.000748*
Somerset County	*59*	*0.000376*	*0.000350*	*0.000203*	*0.000295*	*0.000434*	*0.000705*
Sussex County	*41*	*0.000111*	*0.000107*	*0.000069*	*0.000085*	*0.000133*	*0.000183*
Union County	*104*	*0.001037*	*0.000835*	*0.000409*	*0.000592*	*0.001325*	*0.003171*
Warren County	*24*	*0.000214*	*0.000202*	*0.000068*	*0.000107*	*0.000281*	*0.000570*

Attach.1(8/1/00)
New Jersey - Summary
1990 Modeled Concentration of HAP
arsenic in ug/cubic meter

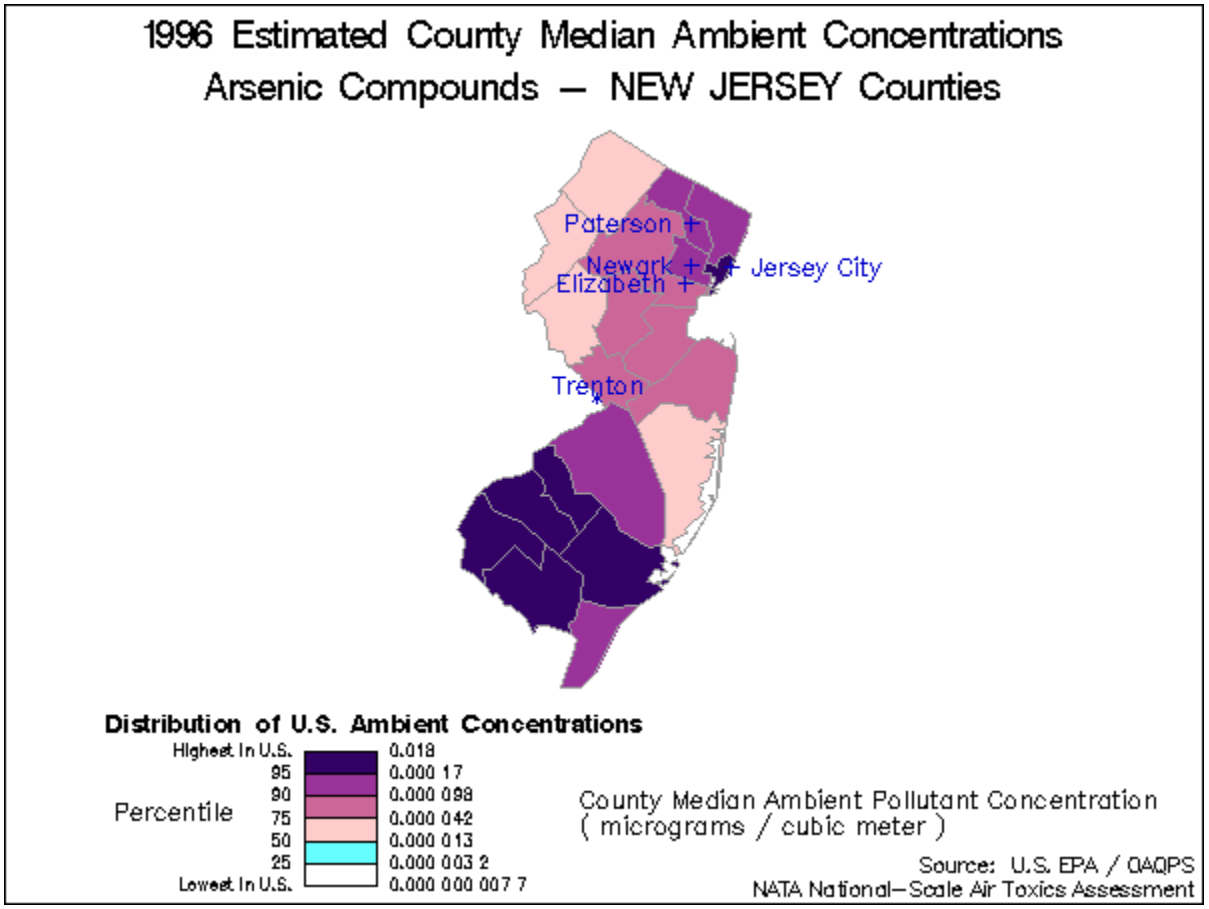
1 of 1 1218/98 2:39 PM
Attachment 2
http://www.epa.gov/
Technology Transfer Network
National Air Toxics Assessment

http://www.epa.gov/ttn/atw/nata/

National-Scale Air Toxics Assessment
Results: Map of 1996 Modeled Ambient Concentrations

These results have limitations (see below)

Issue: Arsenic
Author: B.B. Salahi
Version: 06/20/01



EPA strongly cautions that these modeling results should not be used to draw conclusions about local concentrations or risk. The results are most meaningful when viewed at the state or national level; for smaller areas, the modeling becomes less certain. In addition, these results represent conditions in *1996* rather than current conditions.

The modeled estimates presented here are not a direct indicator of risk because they do not factor in the extent to which people are exposed to these pollutants or the widely varying toxic potential of different substances. In the next step of this assessment, EPA will use these ambient concentration estimates in combination with exposure modeling and health effects information to estimate risk.

Issue: Arsenic
Author: B.B. Salahi
Version: 06/20/01
The emissions used in this assessment do not reflect potentially significant emission reductions that have taken effect since 1996, including those from: 1) mobile source regulations which are being phased in over time; 2) many of the air toxics regulations EPA has issued for major industrial sources; 3) State or industry initiatives; and 4) any facility closures.

Methods of estimating emissions, as well as simplified modeling assumptions, may introduce significant uncertainties into each component of the assessment. See the full discussion of these limitations.

Because of these uncertainties, EPA will not use the results of this assessment to determine source-specific contributions or to set regulatory requirements. However, EPA expects to use these results to inform decisions about the priorities of the air toxics program as well as to guide the collection of additional data that could lead to regulatory decisions.

This request took 4.98 seconds of real time (v8.0 build 1330).

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(8) 1996 Arsenic Emissions, EPA National Air Toxin Assessment.

(9) Characterization of Ambient Levels of Selected Metals and Other Analytes in New Jersey Soils: Year 1, Urban Piedmont Region, 1997, Presented to NJDEP by BEM Systems, Inc., Florham Park, New Jersey.

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www.epa.gov/ostwater/fishadvice/vol1/doc2ndx.html

Uarsenic5.19.2000.(REV.10/27.00)6.20.01B

Issue: Benzene
Author: Gloria Post
Version: 03/03/01

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Benzene**
Description of stressor (including etiology)	Benzene is an aromatic hydrocarbon. The primary source of benzene is petroleum. It is used industrially both as a solvent and a chemical intermediate. It is added to gasoline to increase the octane rating.
Stressor-specific impacts considered including key impacts	Acute: Like other organic solvents, it causes CNS toxicity . Chronic and subchronic: The hematopoietic system is the primary target for benzene toxicity. Exposure results in bone marrow depression, and aplastic anemia in severe cases. Epidemiological studies have linked exposure to benzene with leukemia in humans.
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	Occupational exposure. Exposure while using gasoline. Inhalation of benzene from air near source of release and/or ambient air. Ingestion of contaminated drinking water. Inhalation and dermal during household use of contaminated water (e.g., showering). Primary and secondary exposure to tobacco smoke. Direct contact with contaminated soil.
Population(s)/ecosystem(s) exposed statewide	The general public is exposed via inhalation when fueling vehicles with gasoline-containing benzene Individuals working at gasoline stations and garages, and in facilities using benzene as a solvent or intermediate, would have higher exposures via inhalation. The general public is exposed via inhalation of ambient air, particularly near highly traveled roads. Benzene has been detected in public water supplies and private wells, resulting in exposure via ingestion and inhalation to individuals using these water sources.
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	Water In sampling of public water supplies from 1993-present, benzene was detected (detection limit 0.5 μg/L) in 18 of approximately 600 public supplies (3%). In 8 of these supplies, benzene levels exceeded the MCL of 1 μg/L. The highest level detected was 20 μg/L, while all other systems were less than 5 μg/L. The population served by the

	supplies in which benzene was detected was approximately 296,000, with about 180,000 served by supplies in which benzene exceeded the MCL. Benzene was also detected in 19 non-community water supplies, serving about 4200 people. In 6 of these supplies serving 1100 people, the MCL was exceeded, and the highest level detected was 28 µg/L. In supplies in which MCLs are exceeded, it is required that corrective action be taken rapidly. Therefore, the exposure to benzene via drinking water does not continue to occur. The USEPA Cumulative Exposure Project estimated 1990 benzene concentrations in New Jersey air. Levels ranged from 1 to 342 times the health benchmark concentration, which is the one in one million lifetime cancer risk level. The average benzene concentration statewide was estimated to be 3.3 ug/m3, which results in a cancer risk of 2.8×10^{-5}. From 1990 to 1997, benzene concentrations in air measured in Camden, NJ decreased by approximately 50%. In USEPA's 1996 National Air Toxics Assessment, average ambient air levels of benzene in New Jersey were reported to be 1.66 ug/m3, corresponding to a cancer risk of 1.4×10^{-5}. This concentration is slightly above the national average of 1.39 ug/m3. The county with the highest average level was Hudson, with 2.95 ug/m3 (corresponding to a risk of 2.5×10^{-5}). The greatest source of benzene in ambient air was onroad vehicles, with nonroad vehicles (e.g. construction equipment) the second highest source. Manufacturing and solvent use contributions were approximately an order of magnitude less than from vehicles.
Specific population(s) at increased risk	Toxicity and carcinogenicity of benzene results from its metabolites, rather than benzene itself. There may be certain subpopulations who are more sensitive or resistant to benzene, as a result of the extent of metabolism. Recent studies of Chinese workers have shown that a mutation in the NAD(p)H:p-quinone oxidoreductase enzyme which reduces quinones to hydrooquinones affects susceptibility to myelotoxicity. Differences in enzymatic rates of benzene hydroxylation also affect susceptibility. Benzene metabolism may be induced by exposure to other organic chemicals. Individuals using water contaminated with benzene or living in areas with elevated benzene concentrations in air are at increased risk compared to others.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	There are no differences in exposure level between groups of different metabolic sensitivity. The exposure levels of people with elevated drinking water levels and air levels are discussed above.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	Inhalation Benzene is classified as a Known Human Carcinogen (USEPA Group A). The unit risk is 2.2×10^{-6} to 7.8×10^{-6} for lifetime exposure to 1 ug/m3 in air (IRIS). Oral Benzene is classified as a Known Human Carcinogen both by USEPA and by New Jersey. The slope factor, based on human epidemiological data, derived by New Jersey which forms the human health basis for the New Jersey drinking

	water standard, is 0.23 (mg/kg/day)-1. The USEPA IRIS slope factor, based on a recent reevaluation of the human data, is 0.015 - 0.055 (mg/kg/day)-1.

Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	Water It is not possible to provide an estimate of the number of cancer cases resulting from elevated benzene in water because the slope factors used to develop such estimates are based on lifetime exposure. When elevated levels of benzene are detected in water, the situation is quickly corrected. Therefore, the exposure is stopped and use of these lifetime parameters is not appropriate. Air The lifetime cancer risk from exposure to average New Jersey ambient air benzene levels is 3.65×10^{-6} to 1.3×10^{-5}. In Hudson County, the county with highest average levels, the risk range is 7.4×10^{-6} to 2.2×10^{-5}. The risk from the reported national ambient air background level alone of benzene is 1.1×10^{-6} to 3.6×10^{-6}.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Acute CNS effects are reversible. These occur at high levels not likely to be relevant to environmental exposures in New Jersey. Chronic Both the non-carcinogenic endpoint, aplastic anemia, and leukemia are severe, persistent, and irreversible. The risks of these effects from environmental exposure in New Jersey is likely to be low.
Size of population(s) affected	
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	There are a number of different risk estimates derived from the same occupational data. The differences between these estimates result from the choice of model used and the exposure estimates used. There is large uncertainty in the actual exposures of the workers in the studies which form the basis for the slope factor. There is also uncertainty about the shape of the dose-response curve for benzene at low doses. There are a number of arguments for and against linearity at low doses. The data on benzene levels in private wells is not complete, as private well monitoring is not required. The certainty of the National Air Toxics data is limited because it is based on modeling assumptions rather than actual measured levels.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	M - Future research will provide epidemiological data with better exposure information, more information on the basis for susceptibility to benzene toxicity, and increased knowledge of the mechanism of benzene toxicity.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , ≂, where + is improvement)	++ - Air concentrations of benzene have decreased in Camden since 1990. NJDEP has a multifaceted approach for decreasing emissions of air toxics, including benzene. The Federal Reformulated Gasoline program has limited the benzene content of gasoline to 1%.

Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	Low

Extent to which risks are currently reduced through in-place regulations and controls	Air emissions of benzene are regulated by facility permits. The content in gasoline is capped at 1%. There are drinking water, surface water, and ground water standards for benzene.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry Plus	H - According to CEP, point sources account for 28% of benzene in air.
Small business industry	H - According to CEP, point sources account for 28% of benzene in air.
Transportation	H - According to CEP, 49% of benzene in air results from mobile sources.
Residential	L - Hobbies and home use of gasoline.
Agriculture	L - Gasoline for farm equipment.
Recreation	L - Gasoline for boats, snowmobiles, etc.
Resource extraction	L
Government	L
Natural sources	M - According to CEP, 15% of benzene in air is from "background."
Contaminated sites	M - from petroleum or industrial use.
Diffuse and non-NJ sources	
Sediment	L - not bioaccumulative
Soil	L

Non-local air sources (including deposition)	L
Biota sinks	L

Issue: Benzene
Author: Gloria Post
Version: 03/03/01

Human Health Issue Summary: Benzene

What is it?
Benzene is a colorless liquid that occurs naturally, but most benzene in the environment is a result of its industrial applications. Major sources are chemicals manufacturing, tobacco smoke, and household products such as glue and cleansers. Benzene is also added to gasoline to increase the octane rating. People become exposed to benzene through inhalation of vapors that are present in the environment at low background levels as well as at elevated levels, particularly at gas stations. Benzene can contaminate ground water drinking supplies, mostly as the result of leaking petroleum storage. Benzene is a human carcinogen. It is also toxic to the liver and central nervous system but these non-cancer effects are uncommon in non-occupational settings.

What's at risk?
The general population is exposed to low, background concentrations and higher levels when fueling vehicles with gasoline containing benzene. Individuals working at service stations and in industrial facilities using benzene would have higher exposures than the general public. Drinking water is a potential source of exposure, but known cases of benzene-contaminated drinking water are quickly addressed.

What are the human health impacts in New Jersey?
Excluding occupational exposures, the general public is exposed to benzene levels that may result in a total of 30 to 109 additional cancer cases in New Jersey. The higher rate reflects exposures in more urban areas. This amounts to between 0.4 and 1.6 additional cases per year attributable to benzene. Non-cancer risks from benzene are likely to be low. This assessment did not focus on indoor exposures which may be significantly higher than exposures to outdoor ambient conditions.

What's being done?
The benzene content of gasoline is regulated, and the use of benzene in consumer products is being phased out. Benzene in drinking water is often monitored in areas where there is a history of contamination.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discreter communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
2	2	2	2

Issue: Cadmium
Author: Robert Hazen
Version: 01/02/01

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Cadmium**
Description of stressor (including etiology)	Cd is a metal (atomic number 48) which together with zinc and mercury belongs to group Iib in the periodic table. In animal tissue it occurs in a protein called metalothionine and is bound to sulfhydral groups. In nature it is found in association with zinc and forms chloride, oxide and sulfide salts. (From World Health Organization Health Criteria document no. 134, 1992).
Stressor-specific impacts considered including key impacts	Long-term accumulation in the kidney resulting in disjunction. Inhalation exposure to cadmium sulfate and cadmium oxide resulted in bronchoalveolar benign and malignant adenomas in rats. Effects in animals and humans include nephrotoxicity, fibrotic changes in the liver, interstitial pneumonitis and emphysema, effects on calcium metabolism resulting in osteoporosis and hypocalcaemia, decreased hemoglobin concentration, elevation of blood pressure, reduced testicular weight, fetal toxicity and teratogenicity. Cadmium is also a human carcinogen by inhalation. (From World Health Organization document no. 134 1992). The kidney effects are the only ones attributed to relatively low level chronic environmental exposure through diet.
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	Human exposure occurs through air, food, and water.
Population(s)/ecosystem(s) exposed statewide	General population exposure mostly through the diet. Only one exceedence of the drinking water standard of 0.005ppm this occurred on February 9, 1999 at the Taylortown Road Treatment Plant, at a level of 0.007ppm. Air concentrations statewide according to the National Air Toxics Program do not exceed 1E10-4 ug/m3.
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	Typical exposure is 50ug, 1ug, and 0.015ug per day respectively in food, air and water. (From Cadmium in the Environment, L. Friberg, 2nd edition, CRC Press 1974).
Specific population(s) at increased risk	Subsistence fisherman. Cadmium accumulates in the hepatopancreas of the blue claw crab (and lobsters). This organ is consumed by a substantial subset of crabbers. Also some people cook whole crabs in spaghetti sauce, therefore liberating the high cadmium concentrations in the hepatopancreas and also in the gut. Crabs ingest sediments as they feed and sediments are the part of the aquatic compartment which accumulates cadmium. Modeling suggests that if Cd is applied on a repeated basis to garden crops in NJ at the U.S.EPA's permissible concentration for Cd in sewage sludge for land application, ~20% of consumers of such crops might exceed the Reference Dose for Cd. (Stern, 1993). However, the current extent of use of sewage sludge for amendment of soil in home gardening in NJ is not known.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	At risk population might have 7 times average exposure. This is based on estimate of 30 micrograms per day typical exposure and 200 micrograms per day in polluted areas of the general environment. (Friberg 1974)

Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	Critical level in kidney cortex is 200 parts per million cadmium. 200 micrograms cadmium ingestion per day, long-term, has resulted in proteinuria (Friberg 1974).
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	NJ population at risk probably limited to subsistence fisherman, although general population exposure is approximately 15% of a critical exposure. Cadmium is a carcinogen by inhalation but air concentrations in New Jersey do not result in a risk greater than 10^{-6}. (National Air toxics data).
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Proteinuria from long-term cadmium exposure is irreversible. However, it is not itself a serious health effect. The extent to which it may be predictive of more serious kidney toxicity is unclear.
Size of population(s) affected	A few hundred subsistence fisherman
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	M.... Dose response and toxic endpoint is fairly well established. Exposure in NJ population is poorly understood.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	M.... Extensive human and animal work with this agent. "Recent data indicate that adverse health effects from cadmium exposure may develop in about 1% of the adult general population at an average daily intake of 30ug over a life-span. In high-risk groups the percentage will be even higher (up to 5%). This intake is already exceeded by some population groups in Europe, and the margin is very narrow for large groups. Therefore, measures should be taken to reduce cadmium exposure in the general population to minimize the risk of adverse health effects. At an average daily intake of 70ug/day (corresponding to the present provisional tolerable weekly intake PTWI, WHO), 7% of the adult general population would be expected to develop cadmium-induced kidney lesions. ...Thus, in our opinion, the current PTWI is unacceptable and need to be lowered." (From Scand J Work Environ Health 1998; 24 suppl, page 8).
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =, where + is improvement)	0 Most Cd exposure is from food (grains) which is not of local origin and for which no substantial change in Cd concentration could be predicted
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L..... not aware of any potential for this.
Extent to which risks are currently reduced through in-place regulations and controls	No regulations on food which is the biggest source of exposure. Air and water sources are small compared to food.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	M Historically contaminated sediment is the primary source. Industrial activities such as battery manufacture, metal plating, battery and pigment production lead to Cd in the sediments.

Small business industry	L
Transportation	L
Residential	L
Agriculture	M Ingestion of food grown on high Cd soil and sludge amended soil.
Recreation	L
Resource extraction	L No mining in NJ for this element.
Government	L
Natural sources	L
Contaminated sites	L
Diffuse and non-NJ sources	Most exposure to the general population is due to grain and organ meats which may originate anywhere.
Sediment	H This would be the only local source of significance because of its contribution to Cd in Blue Claw crab hepatopancreas.
Soil	L
Non-local air sources (including deposition)	L
Biota sinks	H

Human Health Issue Summary: Cadmium

What is it?
Cadmium is a rare, naturally occurring metal found in the atmosphere as a result of volcanic activity, ocean spray, and forest fires. Industrially, cadmium is used in electroplating processes, pigments, batteries, plastics, and alloys. Industrial releases to the air and water results in contamination of soils and sediment. Shellfish ingest sediments as they feed, which may expose humans who consume them to harmful levels. Human exposures can also result from air and drinking water concentrations. Chronic low level exposures may result in kidney damage, and cadmium is a carcinogen when inhaled in sufficient concentrations.

What's at risk?
The general population is exposed to low levels of cadmium present in food. Subpopulations at increased risk include subsistence fishing populations and others who consume whole crabs that may contain elevated levels of cadmium stored in the organs. Increased dietary exposure may also result from repeated applications of sewage sludge to garden crops.

What are the human health impacts in New Jersey?
Background levels to which the general population is exposed (including food, air, and drinking water pathways) are estimated at 30-50 micrograms per day. More than 95% of this

Issue: Cadmium
Author: Robert Hazen
Version: 01/02/01
exposure results from levels of cadmium in food. Changes in kidney function have been observed beginning at 200 micrograms per day. The extent to which these changes are predictive of serious kidney problems is unclear. However, recent research indicates that even at background levels, about 1% of the population may develop adverse health effects. Subsistence shellfishing populations may be exposed to cadmium levels seven times higher than background, placing them over the threshold for changes in kidney function. Air concentrations in New Jersey below the level at which additional cancers might be expected. The extent of use of sewage sludge in home gardening in New Jersey is not known.

What's being done?
Industrial discharges of cadmium to the environment are regulated, and cadmium-contaminated hazardous waste sites are cleaned up in accordance with federal and state law. There are no regulations on food, which is the biggest source of exposure in human populations. Use of cadmium in consumer products is being reduced

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discreter communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
L	M	Y	
1	2	3	
			L 2

Reference:

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Issue: Carbon Monoxide
Author: Ken Jones, Sue Thomas
Version: 04/20/01

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Carbon monoxide (CO)**
description of stressor (including etiology)	Carbon monoxide is a by-product of combustion. The compound is formed during combustion of carbon- containing fuels with limiting oxygen. Therefore, one of the conditions to reduce carbon monoxide generation is to ensure significant oxygen during combustion. Automobile fuels have recently been modified to incorporate oxygen containing compounds such as methyl tertiary butyl ether (MTBE) as a way to reduce carbon monoxide emissions. These fuel additives are particularly effective during the cold weather months when carbon monoxide contamination is most prevalent. Because inefficient combustion is the primary cause for carbon monoxide formation, smaller combustion facilities, including home heating devices are significant sources. Smoking cigarettes is also a significant personal exposure route for carbon monoxide. (Doull et al.)
stressor-specific impacts considered including key impacts	The impacts from carbon monoxide are associated with the chemical's ability to bind to hemoglobin more effectively than the natural ligand, oxygen. (Kleinmann) Therefore, one of the ways to measure the impacts of carbon monoxide is to monitor the percentage of hemoglobin that is bound to carbon monoxide (carboxyhemoglobin). At high levels (50%) of carboxyhemoglobin, asphyxiation occurs resulting in death. At lower concentrations, symptoms consistent with decreased oxygen availability such as angina are present. (Doull et al.)
Exposure Assessment	
exposure routes and pathways considered (include indoor air as appropriate)	The sole route of exposure considered in this analysis is via inhalation. Carbon monoxide exists in both outdoor and indoor air.
population(s)/ecosystem(s) exposed statewide	For this analysis we will consider two populations exposed to outdoor concentrations and two populations exposed to indoor concentrations. For outdoor exposures, a general population is considered that is exposed to the average levels determined at the

	monitoring systems from around the state. A highly exposed sub-population is determined for 3 urban counties (Burlington, Union and Bergen), which have 1% of their readings showing concentrations greater than 2.5 ppm. (US EPA (a)). For indoor exposures, there may be general increased carbon monoxide exposures in homes with gas stoves. EPA estimates that CO concentrations in homes with gas stoves range from 5-15 ppm (US EPA(a)). A smaller subpopulation may be exposed to very high levels as the result of accidents. National statistics suggest that the number requiring medical attention may be about 400 in New Jersey each year with between 4 and 25 deaths (Chimney Safety Institute). These accidents are concentrated among low-income households, often using poorly maintained heating devices and an increase in accidents occurs during power outages.
quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	For outdoor exposures, the general population is exposed to an average concentration of less than 1 ppm carbon monoxide. Occasional single hour peak values may approach 5 ppm but these events are isolated and more than 90% of the time, the exposure is less than 1ppm. For more urban populations, average concentrations fall between 1 and 2 ppm. Peak values may approach 8ppm during single hour periods once per year, but 90% of the time the values are below 2 ppm. As noted above, indoor exposures can be much greater than outdoor exposures. General concentrations of between 5 and 15 ppm are typical in homes with gas heating appliances and poorly maintained appliances can result in concentrations exceeding 30 ppm. During appliance malfunction, accidental exposures result in concentrations causing acute symptoms. (US EPA(a))
specific population(s) at increased risk	Chronic angina sufferers are at risk from carbon monoxide exposure because of angina pain associated with decreased availability of oxygen. In the United States, 2% of the population is believed to suffer from angina (www.mamashealth.com). In New Jersey, that translates to about 165,000 individuals.
quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations)	Assuming that angina sufferers are evenly distributed throughout New Jersey, 35,000 will be in urban counties exposed to >2 ppm several hundred hours per year. This same population may be exposed to concentrations >5ppm once or twice per year. 130,000 will be in less urban counties exposed to >2ppm less than one hundred hours per year. Considering indoor exposures complicates the picture significantly. There is no comprehensive monitoring of carbon monoxide in homes but EPA reports suggest regular concentrations in homes with gas stoves of greater than 5 ppm (US EPA (a)). If this is true, then all angina sufferers with gas stoves would be at increased risks for a much greater period of time than would result from exposures to outdoor air. Accidental exposures that require medical treatment are estimated to occur 10,000 times on a national basis translating to about 400 cases in New Jersey (Chimney Safety Institute).
Dose/Impact-Response Assessment	
quantitative dose/impact-assessment employed for each population considered	Health based regulatory standards (EPA, NAAQS) are based to protect individuals from accumulating 2% levels of COHb (Kleinmann). The accumulation of COHb can either occur during short exposures to higher levels of CO (35 ppm for one hour) or for longer exposures to lower concentrations (eight hours at 9ppm). The 2% COHb level is where clinical studies on decreased time for the onset of angina have been carried out (Kleinmann).

	There are other studies looking at acute exposures and other health endpoints including atherosclerotic heart disease, chronic obstructive lung disease and neurobehavioral effects. These effects are most often observed at COHb levels greater than 5% which is evident in smokers and those with occupational exposures (parking garage and tunnel workers) but not those with exposure to ambient conditions (Doull et al.). Death occurs at COHb levels of 50%. Accidental and intentional carbon monoxide exposures occur as the result of motor vehicle emissions in closed areas or as the result of poorly functioning residential furnaces and hot water heaters.
Risk Characterization risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	While 2 ppm is less than the level identified in most studies, it may be enough of an increase to effect some subset of angina sufferers during specific events or in conditions where other factors (cigarette smoking) may increase susceptibility. For this report, we focus on 2 ppm because it is a level reached in New Jersey and at that level, there will be some measurable increase in COHb, possibly about 0.4%. (Doull et al.) This elevated COHb may have no effect in itself, but added upon other factors, which deplete hemoglobin, there may be a stress on individuals. For indoor and accidental exposures, we can use the estimates determined by the Consumer Product Safety Commission, the American Lung Association and EPA that suggest between 4 and 25 deaths in New Jersey and about 400 cases of immediate medical attention. In addition, using the analysis for outdoor air pollution, essentially all sufferers from angina may face increased risk from indoor exposures, especially when in homes with combustion sources. In New Jersey, less than 5% of owner occupied housing has non-combustion sources (usually electric) for heat and 88% of New Jersey households are in structures with less than 5 units (those where individual heating appliances are most likely in use) (US Census (b,c).
risk estimate(s) by population at risk, cont.	For the less urban population, the effects from carbon monoxide may only occur during the occasional event during the year with elevated CO readings.
risk estimate(s) by population at risk, cont.	For indoor chronic risks, there may be a continual risk for angina sufferers, difficult to isolate from the “normal” incidence of chest pain.
risk estimate(s) by population at risk, cont.	For indoor accidental risks, 4-25 deaths, 300 cases of medical attention.
assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	M - Angina is a painful condition that typically will cause its sufferers to stop their activities and seek rest and medication. Events may last a few minutes or longer if the sufferer continues their activity without medication (nitroglycerin tablets offers short-term relief) (www.mamashealth.com). H/M – For acute exposures due to accident, death may result or medical attention is required. Removal of the patient from CO exposure usually leads to a reduction of symptoms.
size of population(s) affected	
assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps (outdoor)	L - The regulatory standard for carbon monoxide was established based on extensive human studies. Studies prior to 1970 suggested that some of the psychomotor effects may exist at concentrations lower than 3% COHb, but later studies dispute that claim (Kleinmann). However, the studies on angina and other cardiovascular effects have stood up to several years of scrutiny and there is little (if any) controversy about the low concentration effects on angina sufferers.

assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps (indoor)	M- There is a lack of comprehensive data on the levels of CO in indoor environments. There is a possibility that significant populations are exposed to concentrations greater than 9ppm as established under air quality regulations.
potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	M - The characterization of more localized exposures to carbon monoxide, including from indoor sources may provide useful information on exposures.
potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	+ - Outdoor carbon monoxide levels have shown a steady decline over the past 30 years (NJDEP, 1998). Regulations on automobiles and point sources have resulted in a decrease in average carbon monoxide concentrations, decreasing more than 40% nationally since 1986 (US EPA). Additional progress may be difficult to attain without significant additional controls on vehicles, area sources and point sources.
potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	In contained areas, carbon monoxide poisoning claims more than one thousand lives per year throughout the United States (Chimney Safety Institute). Assuming New Jersey is representative of the US, there would be approximately 30 accidental deaths resulting from accidental indoor exposure. There would also be an additional 400 - 1,200 hospital admissions.
extent to which risks are currently reduced through in-place regulations and controls	Regulations exist on carbon monoxide emissions from vehicles and several classes of point sources. Household appliances are constructed to minimize carbon monoxide generation, but poorly maintained burners may cause significant emissions and are not currently the subject of regulation.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
large business/industry	L – Environmental Defense reports that less than 5% of CO emissions come from point sources.
small business industry	L -
Transportation	M – Mobile sources account for 90% of New Jersey outdoor releases.
Residential	L (outdoor) H (indoor)
Agriculture	L
Recreation	L
resource extraction	L

Government	L
natural sources	L
contaminated sites	L
diffuse and non-NJ sources	
Sediment	
Soil	
non-local air sources (including deposition)	L-M – Carbon monoxide is not as mobile a pollutant as are other criteria pollutants.
biota sinks	

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
General population 2-L/M	4-M/H	Yes, populations with other factors contributing to COHb (smokers)	2-L/M
Urban population 3-M	3-M	Yes, urban areas	3-M
Chronic indoor exposures 3-M	2-L/M	?	4-M/H
	3-M	2-L/M	4-M/H

Issue: Carbon Monoxide
Author: Ken Jones, Sue Thomas
Version: 04/20/01

Accidental indoor exposures 5-H			
			4-M/H

Human Health Issue Summary: Carbon Monoxide

What is it?
Carbon monoxide (CO) is a colorless, odorless gas formed as a by product of incomplete combustion. A component of motor vehicle exhaust, as much as 95% of outdoor concentrations may be attributed to vehicle emissions in urban areas. Carbon monoxide may also concentrate indoors as a result of improperly functioning home appliances such as furnaces, water heaters, and gas stoves. When inhaled, carbon monoxide affects the body's ability to bind oxygen to hemoglobin in the blood, depriving the body of oxygen. At low levels of exposure, symptoms associated with decreased oxygen availability may result, for example CO may trigger an attack in angina patients. Extreme exposures can result in asphyxiation and death.

What's at risk?
The general population is exposed to low levels of carbon monoxide in the ambient (outdoor) air. Residents of urbanized areas are exposed to slightly higher levels, as are any individuals spending time in locations with a high concentration of vehicles (e.g., parking garages, traffic congestion). Households with gas appliances may be exposed to concentrations up to 15 times greater than ambient outdoor levels. Elderly residents are at increased risk of congestive heart failure resulting from the effects of CO exposure. The approximately 35,000 angina sufferers in urban New Jersey counties are particularly susceptible to the effects of carbon monoxide at observed levels. Smoking cigarettes increases personal exposure to CO significantly.

What are the human health impacts in New Jersey?
The national health-based standard for carbon monoxide is 9 parts per million (ppm) averaged over an 8-hour period, and 35 ppm maximum over a 1-hour period. Annual averages in New Jersey are in the 1-2 ppm range. About 1% of the time, urban counties may show slightly elevated concentrations, while remaining below the national standard. Health effects at these levels include the aggravation of angina or other conditions that are associated with decreased oxygen availability. About 35,000 urban residents suffer from chronic angina. Carbon monoxide has also been linked to congestive heart failure, especially among the elderly. About 6% of congestive heart failures in urban areas may be associated with elevated CO levels. At very high levels of exposure, CO can be deadly. Based on national estimates, about 400 New Jerseyans require medical attention for CO poisoning each year, with 4-25 deaths resulting. These exposures are generally due to accidental or intentional exposures to vehicle exhaust in enclosed areas, or malfunctioning home appliances.

What's being done?
Carbon monoxide is regulated under the National Ambient Air Quality Standards program. Emissions requirements have resulted in significant improvements over the last 30 years, and maximum recorded levels of CO in New Jersey have remained below the health standard since 1995. Household appliances are constructed to minimize CO generation, but poorly maintained burners may cause significant emissions and are not currently the subject of regulation.

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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Chromium**
Description of stressor (including etiology)	Chromium is a metallic element. The elemental form is produced by electrochemical processes, but does not occur naturally in the environment. In the environment, chromium (Cr) is largely found in two valence states, Cr^{+3} and Cr^{+6}. Cr^{+3} is a trace nutrient necessary for sugar metabolism and insulin regulation. It is used in pigment manufacture, leather tanning, as a vitamin supplement (chromium picolinate), and in production of chrome-steel alloys. Cr^{+6} is rarely found naturally, and has no known biological role. It is produced from Cr^{+3} ores by roasting and extraction with strong base. It is generally found as the chromate ($(CrO_4)^{-2}$) ion or the dichromate ion ($(Cr_2O_7)^{-2}$). Cr^{+6} is generally a strong oxidizer although its bio-reactivity is limited by solubility. It is used in pigment manufacture, production of chromic acid, cement manufacture, pressure treated wood, anti-corrosives, cutting oils, and metal plating. Cr^{+6} is also a common trace constituent of detergents and some soaps.
Stressor-specific impacts considered including key impacts	Although $Cr^{+3,}$ produced intra-cellularly from $Cr^{+6,}$ may be the ultimate source of Cr toxicity, it is generally considered relatively biologically inert because it generally does not readily cross cell membranes. It will not be considered further in this assessment (ATSDR, 1989). Because of its strong oxidizing and corrosive properties direct contact of Cr^{+6} with the skin, upper respiratory tract, or mucous membranes can cause serious lesions including ulcerations and perforations of the nasal septum even at relatively low concentrations given chronic exposure. (U.S.EPA, 1984). Such effects are, in general, only observed in occupational settings where direct contact occurs on a repeated basis or where ongoing exposure to chromate mists occurs.
	Based on detailed observations and long-term follow-up of occupational cohorts, Cr^{+6} has been determined to be a

	definite human carcinogen by inhalation resulting in lower respiratory cancers including lung cancer (U.S.EPA carcinogen category - group A) (U.S.EPA, 1984; 2000). Insufficient data are available on the carcinogenicity of Cr^{+6} by ingestion and Cr^{+6} is classified by EPA as a group D chemical (insufficient data for assessment of carcinogenicity) by the ingestion route of exposure. Cr^{+6} is well known as one of the most common anthropogenic allergens, producing allergic contact dermatitis which tends to be particularly persistent due to the ubiquity of Cr^{+6} in common home and workplace items. Ingestion of Cr^{+6} may also result in a more generalized allergic reaction among some sensitized individuals (Stern et al., 1993).
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	ingestion - drinking water inhalation - mobilization to air (entrainment from contaminated soils and fills). May occur indoors due to re-entrainment of outdoor material transported into homes. dermal - contact with moist contaminated soil or standing water which has leached Cr^{+6} from contaminated soil. Contact with household products containing Cr^{+6} .
Population(s)/ecosystem(s) exposed statewide	Approx. 180 separate sites in and around Jersey City are known where chromate production waste, containing up to several hundreds of parts million of Cr^{+6} , were deposited as a result of the activities of three major production facilities over much of the 20th century. A Medical Screening Study was conducted by the NJDHSS in Jersey City from 1992-3. The study was targeted to those residents who lived within 1-2 blocks of a known Cr^{+6} waste site. Participation was voluntary, and 811 people in this category participated (NJDHSS, 1994). It can, therefore, be estimated that greater than 1,000 and perhaps greater than 2,000 residents of Jersey City had a high exposure potential for the Cr^{+6} at these sites. In the interim, an extensive remediation effort has been undertaken. In residential areas approx. 90% of the sites have undergone remediation. However, Cr^{+6} is highly mobile in the environment and so can potentially migrate from under protective caps and or from, as yet, unknown buried fill. Elevated levels of total Cr were found in household dust and in urine of residents in homes within 1-2 blocks of a known Cr^{+6} waste site in Jersey City (Stern et al., 1998), suggesting indoor exposure. Following remediation of adjacent Cr^{+6} waste sites, levels of Cr in household dust in houses with previously elevated levels were found to return to background levels (Freeman et al., 2000). Cr^{+6} is also found at other waste sites in NJ mostly as a result of metal plating operations, but data indicating either the number of such sites, or the number of people potentially exposed at these sites in unknown.

	One public drinking water supply in NJ (in Camden County) has had exceedances of the Maximum Contaminant Level (MCL) for total Cr. Since 1994, two exceedances of the MCL were reported for this supply, more than two years apart. Thus, it appears that exposure above the MCL was sporadic rather than chronic. Since most forms of Cr^{+3} are relatively insoluble, it is reasonable to assume that the measured total Cr may be Cr^{+6}. This supply serves 50,000 people. Five separate non-community water supplies (serving e.g., public facilities, institutions, etc.) had exceedances of the MCL for total Cr from 1994-1998. These exceedances appear to have been sporadic. No records of the population served are available. No data are available on Cr concentrations in private wells. Levels of total Cr in air were modeled for 1990 for NJ counties under the U.S.EPA's Cumulative Exposure Project (CEP). These predictions are based on reported and assumed emissions levels nationwide and on air dispersion modeling. The largest mean concentration (compiled by census tract) was in Passaic County, and was about 30 times the lowest county mean concentration.(Cape May County). The maximum concentration by county was also predicted for a census tract in Passaic County.
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	Detailed data on populations exposed to Cr on or near waste sites in NJ do not exist. Detailed data on exposure levels resulting from exposure on or near chromate production waste sites in and around Jersey City (Hudson County) do not exist. Limited air sampling outside residences adjacent to known waste sites during 1991 found levels of 0.004-0.01 Fg/m^3 (Lioy et al., 1992). These values are consistent with predicted Cr concentration for Hudson County from the CEP. Dermal exposure to Cr^{+6} in and around waste sites has not been measured. The total Cr concentrations in the Camden County supply during the two reported exceedances of the MCL (100 µg/l) were 110 and 210 µg/l. Assuming the default drinking water intake of 2 L/day, these concentrations predict a daily Cr intake of 220-420 µg/day. For the non-community supplies with exceedances of the MCL, the concentrations ranged from 150-250 µg/l corresponding to a default intake of 300-500µg/l day. County mean air concentrations of total Cr statewide predicted by the EPA's CEP ranged from 0.0001- 0.003 Fg/m^3, with a statewide mean level by county (weighted by census tract per county) of 0.0014 Fg/m^3. The predicted maximum census tract air concentration of total Cr is 0.04 Fg/m^3. (Passaic County). It is not known what fraction of this concentration can be attributed to Cr^{+6}.

Specific population(s) at increased risk	There are no known factors which lead to increased susceptibility to cancer from Cr^{+6}. For allergic contact dermatitis to arise (i.e., for the reaction to be "elicited") with exposure to Cr^{+6}, the individual must have been previously sensitized to Cr. Sensitization is generally considered to require a higher concentration of the allergen than is required for elicitation. It is estimated that approximately 1-2% of the general population is sensitized to Cr. The proportion of sensitized individuals is likely to be much larger among various occupations, particularly those in cement manufacturing and the construction trades.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	Not Applicable.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	Cancer – inhalation Based on analysis of a large occupational cohort, U.S.EPA derived a unit cancer risk (i.e., concentration of Cr^{+6} in air predicted to result in one excess cancer with lifetime exposure)of 1.2×10^{-2} /Fg/m^3 (U.S.EPA, 2000) Non-cancer – ingestion There is insufficient data to evaluate the ingestion carcinogenicity of Cr^{+6}. The U.S.EPA non-cancer Reference Dose (RfD) of 0.003 mg/kg day for ingestion is based on an animal dosing study in which no effects were seen, and this RfD is thus, based on the absence of an effect (U.S.EPA, 2000). Allergic contact dermatitis A study in which subjects with a known allergic sensitivity to Cr^{+6} immersed their arms in Cr^{+6} gave a no-observed-effect-level (NOAEL) of 20 ppm in solution (Fowler et al., 1999).
Risk Characterization	

Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	Cr waste sites (including chromate production waste sites in and around Jersey City) Based on measured levels of total Cr in outdoor air at residences adjacent to known waste sites (0.004-0.007 Fg/m^3), and assuming that this represents only Cr^{+6}, this represents a nominal excess cancer risk for lifetime exposure of 4.8-8.4 x 10^{-5}. No data are available to estimate the (non-cancer) ingestion risk. No data are available to estimate the allergic contact dermatitis risk. However, preliminary analysis of the extractability of Cr^{+6} from chromate waste sites indicates that at least some locations in and around Jersey City, Cr^{+6} levels in solution from standing water on contaminated soils could result in elicitation of allergic contact dermatitis in sensitive individuals. Consumers of drinking water with elevated Cr^{+6} levels Intakes of 220-420 Fg/day, Cr^{+6} in Camden County representing exceedance of the MCL, correspond to a dose to a 70 kg adult of 3.1- 6.0 Fg/kg/day. Given the current RfD of 3 Fg/kg/day, the reported levels in Camden County represent a modest exceedance of the estimated safe exposure level. Likewise, the intakes estimated for the non-community water supplies of 300-500 Fg/day, representing exceedance of the MCL correspond to doses of 4.3-7.1 Fg/kg/day for a 70 kg adult. It should be noted, however, that these excursions above the MCL appear to be sporadic and short-term. In contrast the RfD is based on long-term chronic exposure.
	Inhalation exposure in ambient air If it is assumed that the population-weighted (i.e., census tract weighted) total Cr concentration in ambient air statewide of 0.0014 Fg/m^3 represents only Cr^{+6}, then the corresponding cancer risk for lifetime exposure is 1.7 x 10^{-5}. For the NJ census tract (in Passaic County) which has the highest predicted exposure, the predicted ambient air concentration of 0.04 Fg/m^3. total Cr corresponds (assuming all Cr is Cr^{+6}, and assuming lifetime exposure) to a cancer risk of 4.8 x 10^{-4}. Given the assumption that all predicted Cr concentrations result exclusively from Cr^{+6} These estimates are likely to be conservative.
assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Cancer - severe and persistent, and generally irreversible. Allergic contact dermatitis - mild to moderate severity, persistent, and moderately reversible. Effects from ingestion (water) - mild, short-term and reversible at reported levels of exposure.
size of population(s) affected	Cancer Statewide average exposure - entire NJ population Maximum exposure (single census tract in Passaic County) approx. 4,000 people.

	Jersey City population adjacent to waste sites currently probably < 1,000 people. Allergic contact dermatitis from waste sites - unknown, but assuming <1,000 people currently exposed adjacent to waste sites in Jersey City, and assuming prevalence of Cr sensitivity in the general population . 1%, then <10 people are currently at risk in Jersey City for allergic contact dermatitis. Non-cancer effects from drinking water exceeding the MCL Public supply in Camden County with reported exceedances of the MCL serves approx. 50,000 people. The number of people served by the non-community systems with reported MCL exceedances in unknown.
assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	Cancer - M - Fraction of predicted total Cr concentration which is Cr^{+6} is unknown. Ingestion carcinogenicity potential is also unknown. Allergic contact dermatitis - H - prevalence of sensitivity in the general population, levels of dermal exposure, and number of people potentially dermally exposed to environmental Cr^{+6} are all largely unknown. Non-cancer effects from drinking water exceeding the MCL - H - Given apparent sporadic nature of the exceedances of the MCL and given the uncertain nature of the health effects which may occur with ingestion of Cr^{+6} above the MCL, it is not clear what, if any, the risk is to consumers of water from these sources.
potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	M - measured data on ambient air exposures and speciation of total Cr in air.
potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	+ with complete remediation of Jersey City chromate production waste sites.
potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L - There are no known potential sources for catastrophic releases of Cr.
extent to which risks are currently reduced through in-place regulations and controls	Cr releases to air are controlled under the NJDEP Air Quality Permitting Program. Cr in drinking water is regulated by A280 and Safe Drinking Water regulations. Jersey City sites continue to be remediated.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	

large business/industry	M - plating industry, fossil fuel combustion
small business industry	M - plating industry
transportation	L
residential	L/M - fuel oil combustion, use of Cr^{+6} as an anti-corrosive in large air conditioning systems
agriculture	L
recreation	L
resource extraction	L
government	L/M - fuel oil combustion, use of Cr^{+6} as an anti-corrosive in large air conditioning systems
natural sources	L
contaminated sites	M/H - Jersey City chromate production waste sites
diffuse and non-NJ sources	
sediment	L
soil	L
non-local air sources (including deposition)	M - potential medium and long-range transport.
biota sinks	L

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
cancer - H - 5	H/M - 4	Y - 4	4

allergic dermatitis - M - 3	L/M - 2	Y - 3	3
non-cancer ingestion - L - 2	H/M -3	Y - 3	2
			4

Human Health Issue Summary: Chromium

What is it?
Chromium is a metallic element that exists in the environment in two different valence states, Cr^{+3} and Cr^{+6}. It is in the environment as the result of human processes such as the manufacture of pigments, anti-corrosives, pressure treated wood, chrome steel alloys, and in leather tanning. A strong corrosive agent, Cr^{+6} can cause severe irritation of mucous membranes, skin, and the upper respiratory tract. It is also a prevalent allergen, found in many common home and workplace products. Ongoing, direct exposures to Cr^{+6} generally occur only in occupational settings, however it has also been determined to be a human carcinogen via the inhalation route of exposure.

What's at risk?
It is estimated that 1-2% of the general population is sensitized to chromium, and there are no known factors leading to increased susceptibility to cancer as a result of exposure to chromium. Exposures are elevated for residents adjacent to waste sites known to be contaminated with chromium. Approximately 180 sites in and around Jersey City (Hudson County) were used as disposal sites for chromate production waste. Ecosystems are largely exposed via contaminated sediments near waste sites. Drinking water contamination is isolated and sporadic.

What are the human health impacts in New Jersey?
Based on measured levels of total chromium in outdoor air at residences adjacent to historical disposal sites, the cancer risk was calculated at 4.8-8.4 additional cancers per 100,000 people. The number of people exposed on or near waste sites is unknown, however, most of these sites have subsequently been remediated. Average ambient air concentrations in NJ are estimated to result in a lifetime cancer risk of 1.7 in 100,000 people, corresponding to 2 excess cancers per year. In the county with the highest estimated ambient air chromium levels the risk is estimated to be 28 times the overall New Jersey average. Occasional exceedances (two incidents in the past six years) of drinking water standards have temporarily exposed tens of thousands of individuals to concentrations exceeding reference doses for short periods of time. However, these results are based on the assumption that all ambient chromium is in the carcinogenic (Cr^{+6}) form, this is likely to result in a large overestimation of risk.

What's being done?
Waste site clean up is slowly reducing the number of sites with known chromate contamination. Drinking water is regularly monitored to ensure that chromium contamination events are infrequent and not severe.

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Issue: Chromium
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Version: 01/28/00

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Issue: Cryptosporidium
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New Jersey Comparitive Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Cryptosporidium** parvum oocysts (Crypto) in drinking and recreational waters.
Description of stressor (including etiology)	Cryptosporidium is an intestinal parasite. It causes self-limiting gastrointestinal illness in most people, but life-threatening illness in persons with weakened immune systems. Oocysts of this parasite are present in all surface waters. They are derived from the feces of infected humans and animals. They are resistant to most chemical disinfectants used during water treatment. (1)
Stressor-specific impacts considered including key impacts	For people with normal immune systems: infection without illness or else gastrointestinal illness, diarrhea, sometimes with nausea, cramps and vomiting. Resolution of symptoms with a few days to a week or more, infrequently requiring hospitalization. For persons with weakened immune systems: severe, prolonged (weeks to months) gastrointestinal illness, unrelenting cholera-like diarrhea, potentially life-threatening. (1)
Exposure Assessment Exposure routes and pathways considered (include indoor air as appropriate) Population(s)/ecosystem(s) exposed statewide	 Major: waterborne (fecal-oral route). Water contaminated with feces of infected humans and animals. Contaminated drinking water and incidental ingestion during swimming/water sport activities. Major: person-to-person fecal-oral transmission can occur in recreational waters and swimming pools, sex practices that brings one into oral contact with feces of an infected person and *via* day-care and hospital-acquired transmission. Minor: At least one food borne outbreak has been documented (fecally contaminated apples used to make cider), and animal-to-human transmission has been established (calves). (1) Potential drinking water exposure: Approximately 3 million persons in NJ use drinking water derived from surface water sources and an unknown but probably small number of people use fecally-contaminated ground water sources (private wells and public non-community water systems such as those existing in some schools, factories, restaurants, and rest stops).

	Potential recreational exposure: 1 million swimmers at NJ state parks (2); Estimated 8 million swimming or engaging in water sport activities in marine or fresh waters (including swimming pools and water parks).
Quantification of exposure levels statewide (include indoor air as separate category as appropriate)	For recreational water: The arithmetic mean source water concentration from two NJ drinking water studies are 0.805 oocysts/liter, after correcting for average 35% method recovery efficiency and 69% oocysts with potentially viable morphologies (3) and 0.265 oocysts/liter, after correcting for average 18% method recovery efficiency and 25% oocysts with potentially viable morphologies (4). The average of the two mean values = 0.535 oocysts/liter. The maximum observed (3) or estimated (4,5) source water concentrations in these two studies was 28 oocysts/liter, after correcting for average 35% method recovery efficiency (100% of the oocysts in this sample had potentially a viable morphology)(3) and 15 oocysts/liter, after correcting for average 18% method recovery efficiency and 25% oocysts with potentially viable morphologies, respectively (4,5). For drinking water: All public water suppliers that use surface source waters filter their water. Concentrations of Crypto in filtered waters are too low to be reliably monitored with existing methods. Concentrations in filtered water can be estimated, however. The average amount of Crypto removal by filtration equals 3.78 logs, or a 6,026-fold reduction, with a range of 2.24 to 5.16 logs, or a 174 to 144,544 - fold reduction (both during summer) based on a laboratory study (6). This average removal (3 different types of filter media were studied) was 3.13 logs for the filters themselves, plus an average empty column removal of 0.65 logs (6). The average source water concentration of 0.535 oocysts/liter. the average filtration removal amount of 6,026 = an average concentration of 8.88×10^{-5} oocysts per liter after filtration. No sensitive sub-populations are known to have above-average exposure levels (excepting perhaps some occupational groups).
Specific population(s) at increased risk (because of the consequences of exposure)	Persons with weakened immune systems (persons with AIDS, bone marrow and organ transplant patients, cancer therapy patients, persons on high-dose steroid therapy, persons with inherited immune dysfunction syndromes).
Quantification of exposure levels to population(s) at increased risk (due to factors other than exposure) (include indoor air as separate category as appropriate)	Same as statewide exposure levels.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for	Human feeding studies of Du Pont *et al*. (7) for one strain of *Cryptosporidium parvum* ("iowa" strain) showed that

each population considered	62% (18) of 29 healthy adult subjects exposed to doses ranging from 30 to 1,000,000 oocysts became infected (shedding oocysts in feces; with or without illness symptoms). Of the 18 infected individuals, 7 (39%) showed symptoms of cryptosporidiosis. The dose-response data were fitted by Haas *et al.* to an exponential dose-response model (8). The model predicts that a dose of 1 oocyst will cause infection in 1 out of every 239 exposed people. Thus, for a risk level of < 1 infected person per 10,000 exposed persons per year, the concentration of *C. parvum* oocysts in the treated water cannot exceed 3.27×10^{-5} oocysts per liter. Note: Du Pont *et al.* have since tested 2 other strains of *C. parvum* and found that infectivity of these three strains varies by 2 orders of magnitude (9). The number of oocysts infectious for 50% of a test population (ID_{50}), for the three strains are: strain 1 ("Iowa"), 132; strain 2 ("UCT"), 1,020; and strain 3 ("TAMU"), 10 oocysts. Haas *et al.* have not published dose-response models for strains 2 and 3. I have calculated approximate infection rates of 1 out of every 1475 and 15 exposed persons for these 2 strains respectively using the exponential dose-response model (10). Impact (rates of infection) thought to be the same for sensitive sub-populations as for the general population.
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	For drinking water: The overall risk is thought to be low (11). One published estimate calculated a total of 1.5 million infections and 643,000 illnesses in the US per year under typical existing treatment; an average of 2.5 logs of Crypto removal (12). NJ's "share" of this estimate (8 million/270 million people; 2000 census estimates) would be 44,400 infections and 19,000 illnesses. There are numerous, large uncertainties in this estimate. These estimates are likely biased upward because they were done to support cost-benefit calculations in support of a new federal drinking water regulation. Another estimate based on NJ source water data, and using an improved data analysis methodology (13), calculated a much lower total of 815 infections, including 317 with cryptosporidiosis symptoms, per year for the estimated 3,000,000 NJ citizens drinking waters derived from surface sources (14). A parallel calculation using the yearly maximum concentration, with the same filtration removal amount, estimated a total of 63 - 117 infections, including 24 - 45 illnesses, for the 3,000,000 citizens on the maximum concentration days (15). There remains several, potentially large uncertainties in these estimates (16). There have been no documented drinking water-related disease outbreaks due to Crypto in NJ since 1976 (19), the year when this organism was first found to be associated with adverse effects in humans (1). However, the amount of endemic disease due to the presence of this organism in treated drinking water is not known. Between 1991-1996, of 77 documented drinking water outbreaks in the U.S. due to infectious disease agents affecting 2 or more persons, 8 (10%) were attributed to Crypto (19). No outbreaks were caused by Crypto in the last published reporting period of 1995-1996 (19), but one outbreak (in Austin, TX) occurred in 1998.

Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	For recreational waters: Dose-response modeling has not been attempted as the amount and frequency of water inadvertently ingested during swimming or water sport activities is not known. There has been one documented recreational water-related disease outbreak due to Cryptosporidium in NJ (20). The outbreak occurred in late July-early August, 1994 at Lake Nummy in the Belleplain State Forest in Cape May County. The number of infected people was estimated at 135 (2/3 under the age of 15). Between 1991-1996, of 68 documented recreational water outbreaks in the U.S., 14 (21%) have attributed to Crypto (19). 27% (6/22) of recreational waterborne disease outbreaks in the US in 1995-1996 were caused by Crypto (19). The majority of these outbreaks occurred in chlorinated swimming or wave pools, indicating that the mode of transmission is likely to be person-to-person via fecal accidents (19).
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	General population = low severity; short persistence; reversible symptoms; infrequent occurrence. Sensitive population = medium to high severity; prolonged duration; symptoms not always reversible (i.e., death); infrequent occurrence.
Size of population(s) affected	General population: Drinking water = 3 million. Swimming/water sport activity = 6 million + 1 million tourists. Sensitive population: Drinking water Swimming/water sport activity
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	High. (see ref. 16). Currently used Crypto detection assay cannot distinguish live from dead organisms or one strain of Crypto from another. Assay is insensitive. Actual concentrations and distribution of infectious organisms in treated drinking waters and recreational waters are not known.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	High. Better assays in the future may show different levels of infectious organisms in untreated and treated waters. For drinking water: +++ UV treatment appears to be effective in inactivating Crypto in treated water (21). If confirmed for other strains of Crypto, and technical issues related to UV sensors can be resolved, UV may be able to effectively control Crypto in treated drinking waters.
	For recreational water: 0

	Current risk estimate is unknown, but amount of risk is not likely to change in the future. Infection during swimming is often immediate person-to-water-to-person in high bather density water bodies such as swimming pools, and thus not controllable due to insufficient time for filtration and disinfection (if any) to remove the organisms from the water.
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	For drinking water: Low, but possible. Of the 8 drinking water-related disease outbreaks due to Crypto in the US between 1991-1996, one affected 3,000 people and another, which occurred in 1993 in Milwaukee, WI, affected an estimated 403,000 people (approx. 50% of the city population)(19). Of the 403,000 infected, approximately 4,000 were hospitalized and approximately 50-100 persons with weakened immune systems died as a result of the outbreak (22). For recreational water: Low, but higher than for drinking water. Of the 14 recreational water-related disease outbreaks due to Crypto between 1991-1996, 4 affected multiple hundreds of individuals (19).
Extent to which risks are currently reduced through in-place regulations and controls	For drinking water: The EPA Interim Enhanced Surface Water Treatment Rule (23), which covers CWS serving 10,000 or more persons, requires a 2-log reduction of Crypto between the source water and the first customer. Monitoring is not required. A "treatment credit" of a 2 -log reduction of Crypto is assumed if certain turbidity requirements are met. A proposed rule covering smaller systems is scheduled for February, 2000 (24). Crypto can only be further reduced by implementing very expensive treatment such as membrane filtration (25). For recreational water: The EPA Source Water Assessment Program Rule (26) includes Crypto as a parameter which must be taken into account in all drinking water source water assessment plans. Some of these waters may also serve as recreational waters. Otherwise, Crypto is not regulated.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	L. Sanitary wastes (see residential)
Small business industry	L. Sanitary wastes (see residential)

Transportation	L. Sanitary wastes. Some boat waste sources. Sanitary wastes from trains, buses, planes, and roadside rest stops.
Residential	Medium. Septic systems are a potential source if improperly sited too close to drinking water wells and possibly recreational lakes as well. Sewage treatment plant effluents contain low concentrations. This source partially supplies river sediment sinks. Periodic medium to high concentrations at some sites during malfunctions or if plant overloaded during and after rainfall events. Some pets (dogs) may be a source of Crypto in storm water.
Agriculture	Medium? Wastes from young calves have been implicated as a source in some waterborne outbreaks, but the relative concentration of infectious oocysts from livestock, vis-a-vis that from humans and indigenous animals in environmental waters, is not known. Bovine fecal wastes may be a source for soil and river sediment sinks via direct inputs and storm water runoff.
Recreation	Medium. Infection during swimming/water sport activities.
Resource extraction	None.
Government	None.
Natural sources	Low. Wastes from indigenous animals have not been linked to human outbreaks, but over 100 species of animals are known to harbor *Cryptosporidium parvum*, the species that is infectious for humans as detected by the current assay. Indigenous animals are a source of soil and river sediment sinks via direct inputs and storm water runoff.
Contaminated sites	None.
Diffuse and non-NJ sources	
Sediment	Low, most of the time. Possibly significant during and following rainfall/run-off events.

Soil	Low, most of the time. Possibly significant during and following rainfall/run-off events.
Non-local air sources (including deposition)	None.
Biota sinks	High. Biota (over 100 animal species) are the source of all *Cryptosporidium parvum* oocysts.

Human Health Issue Summary:Cryptosporidium

What is it?
Cryptosporidum is an intestinal parasite that infects humans and animals. Egg stage organisms are excreted in the feces of infected individuals and are found in virtually all lakes, rivers, and streams. Able to resist most forms of chemical disinfection, large numbers of *Cryptosporidium* in public drinking water supplies caused widespread illness in the City of Milwaukee in 1993.

What's at risk?
Three million of New Jersey's eight million residents get their drinking water from surface water sources that could potentially be contaminated with harmful levels of *Cryptosporidium*. People may also become exposed while swimming, or coming in contact with the feces of infected individuals.

What are the human health impacts in New Jersey?
There have been no confirmed reports of outbreaks due to drinking water in New Jersey since 1976. However, it is difficult to estimate how many people are affected by a waterborne illness because not everyone exposed will develop symptoms and many cases go unreported. In healthy populations, the increased number of cases of minor gastrointestinal illness may be as many as 19,000 or as few as 300 per year. Estimates for the subpopulation of immune-compromised people range from less than 1 death per year to a high-end estimate of 5-10 additional deaths per year. There was a single documented case of *Cryptosporidium* infection from recreational bathing in New Jersey in 1994 with 135 cases reported.

What's being done?
All public water supplies in New Jersey are filtered; filtration results in a significant reduction in the number of organisms, to an average concentration of below 0.0001 organism per liter. Drinking water treatment technologies exist that would provide further protection, but these are not likely to be employed on a widespread basis because of the high costs involved. *Cryptosporidium* is not regulated in waters used for recreational purposes, except where they also serve as sources of drinking water.

Severity of specified health effects at current levels of exposure (H,M,L)	Size of population at significant risk for each health effect (H,M,L)	Are there discrete communities at elevated risk? (Y,N)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L)

Issue: Cryptosporidium
Author: Tom Atherholt
Version: 08/28/00

For drinking water:			
L, 1	L, 1	Y	L, 1
For recreational water:			
L, 1	L, 1	Y	L, 1
			L, 1

Issue: Cryptosporidium
Author: Tom Atherholt
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15. Maximum observed (3) or estimated (4,5) source water concentrations are 28 oocysts/liter and 15 oocysts/liter respectively. Average filtration Crypto removals = 3.78 logs. 15-28 oocysts/liter. 6,026 = 2.5 - 4.7 x 10^{-3} oocysts per liter after filtration. The Haas model predicts that 1.19 x 10^{-2} oocysts per liter = 1 infection per 10,000 exposed people per

day. 2.5 - 4.7 x 10^{-3} oocysts per liter = 0.21- 0.39 infections/10,000/day or 63 - 117 infections, and 24 - 45 illnesses, per 3,000,000 NJ consumers.

16. The Cryptosporidium dose-response model is based on only one strain of Crypto. The human feeding studies were performed, by necessity, on healthy adults alone. The sensitivity of other sub-populations (e.g., children, elderly, pregnant women, or persons with weakened immune systems were tested) vis-a-vis healthy adults is not known. Additional human feeding studies have shown 10-fold differences in human infectivity for three different strains of Crypto among healthy adults (9,10). There are many genetically distinct strains in environmental waters but the genetic analysis of strains from infected humans shows much less heterogeneity (17) indicating that many organisms detected by the Crypto assay may not be infectious. In the environment there are infectious and non-infectious organisms mixed together and the current detection assay can't discriminate one from the other. Nor can it distinguish one strain of Cryptosporidium from another. Also, prior exposure to a given strain of C. parvum results in partial immunity (18). This fact probably warrants a different, more complicated dose-response modeling for environmental exposures.

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Issue: Dioxins/Furans
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Version: 05/04/01

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	**Dioxins/Furans**
Stressor	
Description of stressor (including etiology)	There are several structurally similar dioxins and furans that result from incomplete combustion of complex organic material in the presence of chlorine or in the manufacture of paper or pesticides and herbicides. There may be small amounts of dioxins and furans formed during natural fire of forested land. A focus of interest in the potential carcinogenicity and toxicity of dioxins and furans was sparked because of the use of defoliants (such as Agent Orange) in the Vietnam war that contained trace contaminants of dioxins, and the potential health effects that resulted in soldiers exposed during that time.
Stressor-specific impacts considered including key impacts	Dioxins and furans are listed as known human carcinogens (National Toxicology Program). In addition, there are other possible effects. At high concentrations, severe acne (chloracne) is evident, and it is possible that immune function and neurological development are affected. At a biological level, there is an induction of increased levels of certain enzymes and altered cellular function for dioxin concentrations slightly above current normal body burdens. The results of these biological changes are uncertain but may include developmental changes, endocrine disruption, and susceptibility to disease. (US EPA, 1985) This report will not cover the possible hormonal effects of dioxins and furans as those effects are discussed in a separate paper.
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	The primary route of exposure for humans is ingestion. (US EPA, 2000). Very little dioxin is airborne, and the compound is not water-soluble so that its presence in drinking water is negligible. Dioxins are fat-soluble and bioaccumulate so they are most often found in animal fats including milk.
Population(s)/ecosystem(s) exposed statewide	Dioxins and furans are present in many food products, so that all humans are exposed. Fat samples from humans around the world consistently find measurable dioxins. In fact, one of the difficulties in studying dioxin's effects is the general consistency in exposures among populations. It is difficult to find populations with elevated concentrations and more difficult to find populations with significantly lower body burdens (NJ DEP, 1985). However high exposure individuals are possible, because crabs and lobsters

	in the NY/NJ Harbor Estuary, particularly in Newark Bay have quite high levels of dioxins due, at least in part, to contamination from the Diamond Alkali site (Bopp et al.). Some members of the population in and around Newark Bay consume these crabs even though it is technically illegal to do so.
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	Because of its general presence in foods, and because New Jersey receives food from around the country and the world, national-level studies are a useful starting point for considering New Jersey exposures. The standard method for discussing exposure of dioxins is in daily intake levels measured as picograms/kilogram body weight/day (pg/kg/day). Current reports suggest that the average US citizen takes in about 25 pg per day or about .3 pg/kg/day. (US EPA, 2000) As an example, a common source of dioxin is in cow's milk which averages about .8 pg/g of lipid (Lorber et al.). Consuming half a liter (about one pint) of whole milk will result in about 15 pg. However, a 15 kg child drinking that same half liter of milk is exposed to 1 pg/kg/day. While there are many dioxins and furans, their mode of action appears to be similar, and the sum of dioxins and furans can be considered together given equivalency factors to weight the relative reactivity. In this report, all figures are given in dioxin equivalents, where each congener is weighted to approximate the impact equal to the most potent dioxin; 2,3,7,8 tetrachlorodibenzo-*p*-dioxin (TCDD).
Specific population(s) at increased risk	For the purpose of this analysis, we will consider two populations. A general population that ingests about 0.3 pg/kg/day of dioxin and more highly exposed populations that ingest locally contaminated shellfish.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations)	In addition to the intake numbers discussed above, there is also information about actual body burden of dioxin. Typical body burdens are measured as a fraction of lipid and estimates by EPA for the late 1990s suggest average levels of 25 parts per trillion. Certain individuals with greater animal fat intakes may have levels about three times the average (US EPA, 2000). There are no results showing increased body burdens among individuals near contaminated sites consistent with the general belief that food ingestion is the primary mode of exposure. For human populations eating contaminated fish and shellfish, the contamination by dioxins varies across species. In a 1997 study (NYS DEC, 1997) fish concentrations of dioxin range from about 0.5 part per trillion (ppt) to 20 ppt. However, in EPA's evaluation of dioxin toxicity, one conclusion is that continued exploration of potentially exposed persons may identify important cases of elevated exposures. (US EPA, 2000). One possible example of these highly exposed individuals are those that ingest blue crabs or lobsters from contaminated sites and do not separate the hepatopancreas from the muscle tissue. While most fish and shellfish tissue have less than 5 ppt dioxins, the hepatopancreas of blue crab and lobster average greater than 100 ppt and can reach values as high as 250 ppt (NYS DEC, NJ DEP). Eating just 10 grams of the hepatopancreas can result in dioxin intake of more than 1000 pg; 40 times the average American intake.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment	Certain laboratory animals are very sensitive to dioxins effects. Guinea pigs develop tumors at very low concentrations of dioxin.

employed for each population considered	However, hamsters require more than 1,000 times the amount of dioxin to exhibit elevated cancer rates. Epidemiological studies provide additional, but unclear evidence. The most often cited review was performed by Fingerhut in a retrospective study of chemical workers exposed to dioxin (Fingerhut et al.). The results of that study are consistent with the possibility that low levels of dioxin result in increased cancer incidence. However, the study of chemical workers includes a confounding factor because of the extensive exposure to other possible cancer-causing chemicals. Studies looking at mechanistic factors suggest that humans do not have the cellular receptors to allow for very low exposures to exhibit the observed effects suggested by animal studies (the susceptible laboratory animals do have the cellular receptor) (US EPA, 2000). The summary of dose-impact studies from the 1980s was the conclusion that 1 pg/kg/day results in an increase in lifetime cancer risk of 200 cases per million population (US EPA, 1985). For this reason, many regulations were based on permitting increases in dioxin exposure of .005 pg/kg/day yielding an increased lifetime cancer risk of 1 per million. The current re-evaluation of dioxin risks suggests a small increase in the possible exposure allowed, but also notes that current exposures may be in the range to observe health impacts (US EPA, 2000). The evidence supporting non-cancer risks is less substantial. The absence of well studied high exposure individuals makes conclusions about developmental difficulties tentative at best. The only certain results are the incidence of chloracne to individuals exposed to very high levels (Doull et al.). Since the discontinued use of pesticides with dioxin contamination, these levels are no longer relevant in environmental exposure.
Risk Characterization Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	At average levels of ingestion of 25 pg/day, the average New Jersey citizen faces an increased cancer risk of between 1.8 e-3 and 1.8 e-4. This translates to an increased population risk for New Jersey of between 1,400 and 14,000 increased cancers over a lifetime or between 20 and 200 increased cancers per year. As with all risk estimates, these numbers are not predictions of increased cancer. In the language of a 2000 EPA dioxin summary: " 'True' risks are not likely to exceed this value, are likely to be less, and may even be zero for some members of the population." (US EPA, 2000) For the highly exposed sub-population subsisting on crab and lobster from Newark-New York harbor, the individual risk may be 40 times the average risk. This translates to individual risks ranging from 6.4 e-2 to 6.4 e-3.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Cancer from exposure to dioxin is representative of the range of cancers suffered from a combination of causes in the United States. Between a third and 95% of cancer cases result in death and non-terminal cases often require prolonged treatment and significant suffering (Doll and Peto).
Size of population(s) affected	Dioxin is generally distributed and the exposure is considered uniform for all citizens in New Jersey.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	M - Dioxin provides a dilemma in the discussion of uncertainties. It may be the most carefully studied environmental pollutant in the United States, but those studies have raised several important questions and elevated the discussion of uncertainty. Unlike most chemicals, the mode of initial biological reactivity is fairly well understood, but the consequences of dioxin binding to cellular receptors is unclear (Roberts).

	On the exposure side, it is clear that most, if not all Americans have significant body burdens from dioxin exposure, but it is unclear if there are some Americans with significantly increased body burdens. The uncertainty with respect to exposure is elevated with dioxins because of the very small amounts of the compound that are necessary to initiate biological changes. Picogram exposures are much more difficult to ascertain than are microgram or milligram exposures typical for most pollutants. The remaining uncertainty with respect to dioxin and cancer is similar to other chemicals and their role in cancer. Epidemiological studies can never separate dioxin's effects from other chemical exposures and the differences in genetic makeup of humans makes generalizations impossible.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	M - There will remain a continued focus on dioxin and as a result, there is a chance that further mechanistic studies will show certain effects. In addition, further work with potentially exposed populations may identify individuals with higher exposures.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	+ Studies of sediments show that the deposition of dioxins and furans in the environment peaked during the 1960s when the manufacture and use of dioxin-contaminated pesticides was at its height. Since that time dioxin deposition rates have decreased between 50 and 90 percent. (US EPA, 2000). EPA estimates reductions on the order of 80% between 1987 and 1995 with additional reductions likely due to regulation of municipal and medical waste incinerators. However, dioxin is a persistent contaminant and the return to background levels of exposure may require several decades. (US EPA, 2000)
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L/M – Dioxins and furans have been released during industrial accidents. The most famous of these events took place in Seveso, Italy and resulted in the evacuation of several square kilometers. However, there were no direct effects noted except chloracne among a handful of workers (Marrochi et al., Spagnolo et al.). Other exposures such as Times Beach, Missouri have also resulted in evacuation but no increased acute symptoms. As the result of these events, the consequences for a New Jersey accident should be minimal.
Extent to which risks are currently reduced through in-place regulations and controls	Dioxin is the focus for regulations of many combustion sources and power plants. While establishing facility specific emission limits is difficult, public pressure typically results in dioxin control strategies.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	M A few major sources continue to operate in New Jersey, although no individual source is expected to add more than 5% of the total state load (US EPA, CEP).
Small business industry	L
Transportation	M Diesel trucks emit significant dioxin loads (Gullett and Ryan)
	M Incineration of household hazardous waste is a significant contributor to dioxin emissions

Residential	
Agriculture	L
Recreation	L
Resource extraction	L
Government	L
Natural sources	L
Contaminated sites	L/M
Diffuse and non-NJ sources	
Sediment	M
Soil	M
Non-local air sources (including deposition)	L/M
Biota sinks	M

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
Cancer 5-H	4-M/H	3-M	4-M/H
Non cancer 2-L/M	4-M/H	3-M	

			2-L/M
Subsistence fishing 5-H	3-M	5-H	4-M/H
			4-M/H

Human Health Issue Summary: Dioxin

What is it?
Dioxins (and furans—for the purpose of this analysis, they have been grouped together) are a group of structurally similar chlorinated compounds that result from the combustion of complex organic material in the presence of chlorine. These compounds may also arise as by-products of paper production or the synthesis of certain pesticides. These trace contaminants are biologically active at very low concentrations and accumulate in soils and sediments via air and wastewater releases. Aquatic life feeding on sediment- dwelling organisms accumulate dioxin in their tissues and terrestrial organisms become exposed by feeding on aquatic organisms or other terrestrial species (including plants) that have taken up dioxin from the soil.

What's at risk?
Because dioxin is ubiquitous in our environment, all species are exposed. Animals higher on the food chain can be exposed to greater quantities as a result of bioaccumulation in the environment. For humans, the primary sources of dioxin are meat, fish, and dairy products. Individuals may be exposed to high levels of dioxin when contaminated shellfish are a significant part of the diet. Dioxin is a carcinogen and also affects other biological functions such as the reproductive system of many species.

What are the human health impacts in New Jersey?
In the general population, dioxin exposure may contribute to an additional 20-200 cases of cancer per year in New Jersey. Highly exposed individuals such as those who regularly eat contaminated shellfish from Newark harbor may face individual risks that are forty times the general population risk of 1.8 –18 excess cancers per 10,000 population. Non-cancer effects are also possible, but no concrete estimates are available.

What's being done?
Dioxin releases from several types of facilities are regulated, resulting in a steady decrease in emissions. Sites contaminated with dioxins as a result of chemical operations are being identified, isolated from human exposure, and slowly cleaned up. Bans on the consumption of shellfish that is known to be contaminated are intended to reduce the exposure to those for whom shellfish is a subsistence food.

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Issue: Disinfection by-products
Author: Perry Cohn
Version: 01/09/01

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Disinfection By-products**
Description of stressor (including etiology)	Disinfection by-products (DBPs) are a diverse group of chemicals, only about half of which by weight have been chemically characterized, formed by the reaction of chlorine and related chemicals with influent organic material, including naturally occurring humic and fulvic acids from vegetation and algae, during water treatment. Organic material is highest in surface water. The DBPs with the highest concentration include the trihalomethanes (THMs), including chloroform and bromodichloromethane, and the haloacetic acids (HAAs), such as di- and trichloroacetic acids. Other identified DBP chemical families are haloaldehydes, haloketones, haloacetonitriles, chloropicrin, cyanogen chloride, and chlorophenols. Until recently, drinking water standards required only THM measurements and the MCL was a running annual average of less than 100 ppb total THMs. Thus, THMs served as a surrogate for all DBPs, many of whose levels do not correlate with THMs. The current USEPA MCL for total THMs is now 80 ppb. Now HAAs must also be tested and require a running annual average less than 60 ppb total HAAs.
Stressor-specific impacts considered including key impacts	USEPA (1998) has acknowledged an association between DBPs and bladder cancer, as well as, possibly neural tube birth defects (NTDs), possibly other cancers and spontaneous abortions. Two New Jersey case-control studies (Bove et al., 1996; Klotz and Pyrch, 1998) have observed a statistically significant association with NTDs.
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	Tapwater from treatment plants supplied by surface water sources, particularly rivers, is the main source. Exposure routes include ingestion, inhalation (the volatile chemicals like THMs), and dermal (e.g., during immersion in a bathtub).
Population(s)/ecosystem(s) exposed statewide	Approximately 55% of NJ population (4.1 million) are served by water utilities supplied by surface water, with varying levels of DBPs depending on organic content, treatment methods, and transit time in distribution system.
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	About 55% of New Jerseyans are served by community water systems (CWS) using a surface water source. There can be considerable variation of THM and HAA levels on a daily, weekly, monthly, seasonal and yearly basis. During the 1995-1999 period about 25% of people in those systems had annual average THM levels over 60 ppb, while 45% had 40-60 ppb, 10% had 20-40 ppb, and 15% had 10-20 ppb. That corresponds to 1 million, 1.9 million, 500,000 and 700,000 people (all rounded), respectively. People served by groundwater in CWSs and private wells generally have less than 5 ppb THMs.
Specific population(s) at increased risk	Pregnant mothers and their fetuses, or about 0.55 x 115,000 live births/yr. x 1.15 (an estimate of 15% embryonic and fetal loss based on Waller et al. (1998) and other studies in California) = 73,000/yr, where 0.55 is the percent of the NJ

	population supplied by treated surface water. Alternatively, the number of women of childbearing age, 18-35, supplied by surface water in NJ is about one million.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	An estimate of pregnant mothers and their fetuses in systems with different levels of THMs are: > 60 ppb THMs, about 0.25 x 73,000 = 18,000 (rounded); 40-60 ppb, about 0.45 x 73,000 = 33,000; 20-40 ppb, about 0.15 x 73,000 =11,000.
Dose/Impact-Response Assessment Quantitative dose/impact-assessment employed for each population considered	Laboratory studies: Exposure to THMs, chloroform, bromodichloromethane (BDCM), dibromochloromethane (DBCM), or bromoform, has resulted in mouse liver cancers and pre-neoplastic nodules (USEPA IRIS), but objections have been raised to dosing with a corn oil medium. Thus far, aqueous solutions have not resulted in cancers, but BDCM has resulted in increased lower intestinal crypt foci (a precursor of colorectal cancer) with both oil and water (DeAngelo and George, 1999). Oxidative BDCM metabolites result in DNA adduct formation, and it is weakly mutagenic and genotoxic. Dichloroacetic acid in aqueous solution increased hepatocellular carcinomas at 50 mg/L (DeAngelo et al., 1999), but it is equivocally mutagenic or genotoxic. Another chemical, called MX, causes cancer at multiple sites at low doses in rodents and is mutagenic, but occurs at very low levels. A cancer slope has not been adopted for either chemical. Based on USEPA rodent cancer slopes for THMs, a typical THM mixture (60% chloroform, 25% BDCM, 10% DBCM, and 5% bromoform) has a lifetime cancer risk of 6×10^{-5} in the 60-80 ppb range (70 ppb mean) and 4×10^{-5} in the 40-60 ppb range (50 ppb mean). Many of these chemicals also cause liver and kidney problems in laboratory animals exposed to high levels (e.g., THMs, HAAs). Low levels of some brominated DBPs, such as dibromoacetic acid, result in male reproductive problems in laboratory animals, but there is little evidence of birth defects and spontaneous abortion. HAAs, especially monobromo- and monochloroacetic acids, have also been linked by in-vitro assays to neural tube defects. For many DBPs and DBP mixtures in general, however, there are insufficient laboratory data on health effects and, as mentioned, only half of the DBPs, by weight have been chemically identified. Thus, with paucity of data and difficult to interpret data from the laboratory, epidemiology is important. Cancer: Bladder cancer is recognized as a health effect in old and new studies. The old studies, when weighted by number of participants and quality yielded a pooled relative risk of 1.2, most comparing exposure to chlorinated surface water, but one comparing total THMs (Morris et al., 1992). Higher exposure (concentration and duration) corresponded to a pooled relative risk of 1.4. Work by Cantor et al. (1987) was the biggest and best of these pooled studies. They found that duration of residence in a system supplying chlorinated surface water and rate of ingestion were both associated with bladder cancer (OR = 1.8 for 60+ years of 2+ liters daily, and among non-smokers with tap water ingestion above the median, OR = 3.1 for 60+ years). Studies published after that meta-analysis are described next. In Colorado, McGeehin et al. (1993) found that after adjustment for cigarette smoking, tap water consumption, and medical history, years of exposure to chlorinated surface water were significantly associated with risk for bladder cancer, OR = 1.8 (95% CI 1.1-2.9) for more than 30 years compared with no exposure.
Quantitative dose/impact-assessment, cont.	
	The increased bladder cancer risk was similar for males and females and for nonsmokers and smokers. Notably, THM levels, as an available surrogate measure, were not specifically associated. In Ontario (King and Marret, 1996) those exposed to chlorinated surface water for 35+ years had an increased risk of bladder cancer compared with those exposed for less than 10 years (OR = 1.41, 95%CI 1.10-1.81). Those exposed to THMs > 50 ppb for 35+ years had 1.63 times

	the risk of those exposed for less than 10 years (CI = 1.08-2.46). In Iowa (Cantor et al., 1998), the association of bladder cancer with chlorinated surface water was OR = 1.9 among men exposed for 60 years or more and 2.2 among people who ever smoked, but among women and non-smoking men there was no association. In Washington County, Maryland (Freedman et al., 1997), the association was also limited to men (adjusted for smoking among other confounders) and to those who had smoked cigarettes.
Quantitative dose/impact-assessment, cont.	In ever-smokers compared with never-smokers with low exposure, the adjusted ORs for bladder cancer risk with increasing exposure were 1.3, 1.4, 1.4, 1.7, 2.2, 2.8, respectively, for 0, 1-10, 11-20, 21-30, 31-40, > 40 years' exposure duration. THM levels were not part of the analysis in Iowa or Maryland. Some older studies have reported an association of chlorinated surface water with colorectal cancer (reviewed by Morris et al., 1992), and two recent studies are consistent. Notably, a recent prospective study of post-menopausal women in Iowa reported an elevated risk of colon cancer with chloroform exposure (Doyle et al., 1997), while a case-control study also in Iowa (Hildesheim et al., 1998) observed linear dose-response with duration of exposure to chlorinated surface water (OR = 2.6 for 60+ years duration). No other cancers have been adequately investigated, but associations with brain and kidney cancers have been reported. Birth defects: Bove et al. (1995), using a birth registry-based (cross-sectional) case-control study with 81,000 live births found that neural tube defects (occurring in the embryonic structure that becomes the brain and spinal cord) in 56 cases were associated with THMs in a dose-response manner, with >80 ppb associated with an OR of almost 3 (95%CI 2-4.4). Klotz and Pyrch (1999) in a case-control study in New Jersey found that singly occurring neural tube defects (NTDs), versus cases with multiple neural tube problems, were associated with >40 ppb THMs (unadjusted OR = 2.1, 95%CI 1.1-4.0; adjusted OR = 2.3, 95%CI 1.0-5.2) compared to those exposed to <5 ppb. Among those who did not take vitamins during pregnancy, exposure to >40 ppb THMs resulted in greater risk (OR = 2.6, 95%CI 1.2-6.0). This apparent effect modification indicates an increased effect of DBPs at low folate levels. Neither HAAs nor haloacetonitriles were associated.
Quantitative dose/impact-assessment, cont.	Spontaneous abortion: In a prospective cohort study in California, Waller et al. (1998) found increased spontaneous abortion (loss in the first 20 weeks of gestation, excluding early embryonic loss before completion of interview around the 8th week) with exposure to >75 ppb THMs among 5,000 women recruited through a statewide HMO when they called to arrange a first prenatal visit. Women drinking five of more glasses of cold home tap water containing at least 75 ppb THMs experienced an early miscarriage rate of 16% compared with a rate of 9.5% among women drinking less than 5 glasses of cold water or any amount of tap water <75 ppb THMs, yielding an adjusted OR = 1.8 (95%CI 1.1-3.0). However, this increase was seen in only one of several study areas. This association was strongest with BDCM. These results appear to be consistent with the results of birth defect studies since many early miscarriages may be due to severe neural tube defects.
	A retrospective cohort study found an adjusted RR = 1.7 (95%CI 1.1-2.5) for the risk of stillbirth among women exposed to >100 ppb THMs averaged over the entire pregnancy relative to women exposed to <50 ppb (Dodds et al., 1999). No association was observed with congenital anomalies, but only 3% of the comparison group had water with average THMs < 25 ppb.

	A case-control study in North Carolina (Savitz et al., 1995) reported no increased risk of miscarriages with THM levels in the first trimester of pregnancy, but ascertainment of spontaneous abortions in the first trimester may have been weak and their THM dose rate metric was probably biased since intake in their subjects was in general inversely related to THM concentration.
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	Cancer: The USEPA (1998) has acknowledged a population attributable risk (PAR, also known as attributable proportion) of 2-17% (within 95% upper and lower bounds, central estimate of 9%) for bladder cancer among those drinking chlorinated surface water in the five most recent high quality case-control studies (including Cantor et al., 1987). (These studies had residential history, but only two had analysis based on THM levels.) This equates to about 1100-9300 new cases each year in the US, based on 55,000 new incident cases per year, or about 40-350 in New Jersey, based on average annual bladder cancer incidence of about 1,500 among males and about 500 among females from 1992-1996. There may be susceptible populations but results have been inconsistent between studies, such as smokers, and analysis has been limited. Neural tube birth defect: A PAR of 9% can be calculated for women in the Klotz and Pyrch (1999) study drinking water with 40+ ppb THMs (as a surrogate for overall DBP exposure) during neural tube closure. Based on about 50 infants born with neural tube defects annually in NJ and about 40% (0.7 x 0.55; see *quantification of exposure levels statewide,* above) of reproductive age women living in systems with average annual 40+ ppb THMs, about 2 cases per year would be attributed to THMs. If the first trimester occurs in the summer, when THMs tend to be highest, the exposed population could be estimated by including systems where the 75th percentile of THMs was >40 ppb (i.e., where one quarter of the samples, mostly in the 3 summer months, were >40 ppb). If 25% of pregnant women are exposed over the summer, then the number of attributable cases would be: 0.25 x population exposed/total population (3.3M/4M = 0.82) x 50 x 0.09 = 0.9 case). Spontaneous abortion: A PAR of 7% can be calculated for women drinking 5+ glasses of water with 75+ ppb THMs (as a surrogate for overall DBP exposure) in the Waller et al. (1998) study. Assuming that spontaneous abortions before and after interview were equally affected by DBPs are about 15% (10% among those interviewed in the first 20 weeks of gestation and about 5% aborting before the interview) of the 115,000 annual NJ births (adjusted to be 115% = 132,000) and with 15% of NJ exposed to >75 ppb (estimation based on: 13% of the population in the summer, represented roughly by the 75th percentile, see above for rationale; and, representative of the spring and fall, only 2% at the 50th percentile), 0.15 x 0.15 x (132,000) x 0.07 = 210 cases.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Bladder cancer: fatal, somewhat treatable Neural tube birth defect: moderate to high degree of severity, but mostly treatable Spontaneous abortion/stillbirth: fatal
Size of population(s) affected	Bladder cancer: Approximately 0.5x0.55x8 million = 2.2 million (i.e., 50% of people in surface water based systems, or

	25% of the NJ population, are exposed to >50 ppb annual average THMs, as a surrogate based on the Ontario study (King and Marrett, 1996). However, most studies did not analyze cancer incidence based on THM or other DBP levels. Neural tube birth defect: approximately 0.7x0.55x1.5 million women of reproductive age in NJ = 600,000 women of reproductive age (70% of the 55% receiving treated surface water will be exposed to an annual average of >40 ppb THMs (with reference to Klotz and Pyrch, 1999) or at a critical fetal period). Spontaneous abortion: approximately 0.1x0.55x1.5 million = 80,000 women of reproductive age (i.e., 10% of the 55% receiving treated surface water will be exposed to >75 ppb THMs (with reference to Waller et al., 1998) over the first trimester).
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	Cancer: M There are many chemicals that make up DBP mixtures and the proportion and concentration in any water system has a wide range of variation. At least one of these has significant mutagenic and carcinogenic activity using aqueous oral ingestion. Numerous epidemiological studies point to modest, but very consistent risk of bladder cancer and, possibly, rectal cancer. Duration of residence in systems using chlorinated surface water or, at best, total THMs have been used as the surrogate for total DBPs in epidemiological studies thus far. Epidemiological studies on which this estimate is based cannot yield results for specific chemicals like laboratory animal experiments, but there is some consistency between the rectal cancer findings in some studies and the occurrence of preneoplastic colorectal crypts in rats treated with BDCM. In addition, known confounders like age and tobacco use were adjusted in the analysis. Neural tube birth defect: M Many of the comments above apply to this category also. There are only two positive studies out of three in the literature, but in the Klotz and Pyrch case-control study various stratifications meant to decrease misclassification of exposure and effect increased the degree of association. The negative study only had a few controls receiving <25 ppb THMs. Positive findings with spontaneous abortion are viewed as being consistent with positive association with neural tube defects. Spontaneous abortion: M Ascertainment of spontaneous abortions by Waller et al. (1998) was probably sufficiently complete and there was data on filter use, tap water ingestion, showering, smoking, alcohol use, and SES indicators.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	Cancer: M (consistent bladder cancer risk, but future studies may find gene polymorphisms in susceptible populations). The association of brain cancer with chlorinated surface water in Iowa (Cantor et al., 1999) is being re-studied in four Midwestern states. The Canadians are conducting a large, nationwide case-control study of multiple cancer sites. Expanded study of the carcinogenicity of BDCM in rodents may increase the significance of the epidemiological studies on colorectal cancer that observed an association with chlorinated surface water. Neural tube birth defect: H (because there are few studies) Spontaneous abortion: H (because there are few studies)

Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, □, =, where + is improvement)	++ : The recently promulgated federal Long-term Enhanced Surface Water Treatment Rule and the expected successor rule are increasing controls on surface water treatment and lowering the allowable levels of THMs, HAAs and probably other DBPs.
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L
Extent to which risks are currently reduced through in-place regulations and controls	Total THM MCL recently reduced to 80 ppb. MCL for total HAAs recently promulgated, 60 ppb.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	M: as wastewater dissolved organic precursors, and nitrogen and phosphorous (that increase growth of algae and vegetation).
Small business industry	L
Transportation	L
Residential	M: as dissolved organic precursors in urban/suburban run-off
Agriculture	M: as dissolved organic precursors, and nitrogen and phosphorous in run-off
Recreation	L
Resource extraction	L
Government	M: waste water treatment facilities: dissolved organic precursors, and nitrogen and phosphorous (that increase growth of algae and vegetation).
Natural sources	M
Contaminated sites	L
Diffuse and non-NJ sources	
Sediment	M: assuming disturbance during storm event

Soil	M: as a result of run-off
Non-local air sources (including deposition)	L

Biota sinks	L

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
H (endpoints are severe), but odds are only moderately elevated.	H, unless there is a susceptible genetic subpopulation	Y	
5	5	5	
			M - 3: also as a function of association of health effects and exposure

Human Health Issue Summary: Disinfectant By-Products

What is it?
Disinfection by-products (DBPs) are a diverse group of chemicals formed by the reaction of chlorine and related chemicals during the disinfective treatment of surface water. DBPs remain in the drinking water available to the public. DBPs have been linked to bladder and possibly other cancers, neural tube birth defects (such as spina bifida), and miscarriage. The DBPs with the highest concentration include the trihalomethanes (THMs) and the haloacetic acids (HAAs).

What's at risk?
About 55% of the New Jersey population is served by water utilities supplied by surface water, with varying levels of DBPs. Populations at increased risk include pregnant mothers and their fetuses, particularly when their drinking water is derived from treated surface water.

Issue: Disinfection by-products
Author: Perry Cohn
Version: 01/09/01

What are the human health impacts in New Jersey?

Based on population percentages established by US EPA, DBPs may be expected to cause 40-350 cases of bladder cancer, 2 neural tube defects, and 200 miscarriages each year in New Jersey. About 25% of the New Jersey population, or half of people served by surface water based systems, are exposed to THM levels greater than 50 parts per billion (ppb), as compared to people served by private wells, which generally have less than 5 ppb. While the US EPA sets the standard for THM at 80 ppb, studies have linked neural tube defects with THM levels greater than 40 ppb.

What's being done?

The Maximum Contaminant Level (MCL) for total THMs in drinking water was recently reduced to 80 ppb, and an MCL for total HAAs was recently established at 60 ppb.

Issue: Endocrine Disruptors
Author: Tom Ledoux
Version: 02/01

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Endocrine Disruptors** (ED)/ Hormonally Active Agents (HAA) in the environment
Description of the stressor (including etiology)	ED are chemicals that mimic or inhibit the action of natural hormones (80+vertebrate hormones) or alter the normal regulatory function of the immune, nervous, and endocrine systems (EPA, 1997). The National Research Council settled on using the term, HAA, (Hormonally Active Agent) to describe substances that possess hormone-like activity, regardless of the mechanism (NRC, 1999). ED/HAA result from intrinsic properties of chemicals intentionally used in products and/or unintendedly released into the environment that involve single or multiple exposures to chemicals, mainly synthetic, or to naturally occurring endocrine active substances. Here the terms ED, endocrine mimics, endocrine modulation, gender benders, and HAA are used synonymously for the sake of simplicity.
stressor-specific impacts considered including key impacts	Human - adverse reproductive and developmental outcomes, both structural and functional (altered sex-ratio, lactational parameters [shortened lactations compromise the ability of the children to grow and develop optimally], developmental landmarks altered [onset of prepubertal changes and puberty itself, age of menarche]) that can be manifested during any time period, from development through senescence.
Exposure Assessment	
exposure routes and pathways considered (Include indoor air as appropriate)	Exposure may occur through water, food, soil, and air in both residential and occupational settings.
population(s)/ecosystem(s) exposed statewide	Wildlife, especially species relying on aquatic and coastal habitats (fish, amphibians, piscivorous birds, etc.) has been most notably impacted to date. Humans are exposed through a variety of exposure routes, e.g., residential and occupational pathways and through exposures related to contaminated soil, water, and food (eating non-recommended species and/or exceeding fish advisory limits). At least to some extent the entire state population is exposed to EDs.
quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	Quantification of ED toxicants in humans is limited at present to relatively few chemicals and to selected hot spots of exposure and contamination. Further, NJ data on likely highly exposed subpopulations and sensitive populations are minimal, if any.
specific population(s) at increased risk	Pregnant women and young children through all developmental stages are especially sensitive. Nursing young of mothers previously exposed to elevated levels of ED chemicals are likely to have high levels in adipose tissue that

	become mobilized at lactation. Individuals consuming food containing high levels of ED active chemicals associated sometimes with □fad□ diets may be at increased risk. Additionally, individuals who, for cultural or economic reasons, consume elevated quantities of contaminated wildlife in excess of advisory limits are likely to be at elevated risk. These highly exposed subpopulations cannot be defined in most cases based solely on geography.
quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	Populations and subpopulations that may be at increased risk are poorly assessed quantitatively at present.
Dose/Impact-Response Assessment	
quantitative dose/impact-assessment employed for each population considered	Not available at present. Heretofore, toxicology has been primarily concerned with high dose effects. It is only recently that there has been an interest in identification and quantification of low dose effects. Ongoing studies are on several highly exposed subpopulations (e.g., Yusho/Yu Cheng populations exposed to PCBs, and PCDFs) and North Carolina (exposed to PCBs), to determine dose response curves and long-term developmental consequences not only to the exposed population but also their progeny. Starting in the 1940's through the end of 1972, pregnant women threatening miscarriage were often treated with a synthetic estrogen, diethylstilbestrol (DES). DES treatment resulted in increased rates of a rare genital tract cancer in female offspring and high rates of reproductive tract abnormalities in male and female offspring. Investigations are currently underway to determine whether these effects are heritable. The mechanism by which DES is acting across generations as it does is not well understood. Although banned for use in humans in 1971, DES was used as a feed additive for another decade before being banned to increase weight gain in cattle and other livestock. Its illegal use continued as late as 1991 in the US when Switzerland detected DES in American exported beef.
Risk Characterization	
risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	Potential for indeterminate elevated risk for heavily exposed subpopulations and sensitive populations. Comprehensive region specific and subgroup quantitative human dose-response data are lacking in most instances.
assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Effects on heavily exposed subpopulations (hot spots) may be moderate to severe, depending on definition. Effects may range from short-term to lifetime depending on agents involved and exposure parameters (developmental stage, concentration, time, etc.). Individuals exposed during sensitive stages of development stages may experience permanent developmental deficits whose degree may range from mild to severe. Some individuals may or may not be aware of slight to some mild forms of deficits, whereas, they would likely be aware of moderate to severe deficits. That being said, not all affected individuals will necessarily learn or become aware or understand the causality of their deficits.
size of population(s) affected	While unknown at present, population affected is likely to be small at high levels of exposure, small to moderate at

	moderate exposure levels, and small, if any, at low levels of exposure. Virtually everyone has been exposed to some degree. This is particularly the case for phthalates which, as plasticizers are ubiquitous, and which appear to have some ED activity. The degree to which young people (who were probably exposed at some indeterminate degree during development) have been affected is problematic at this stage of research.
assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	Uncertainties are high (H) mostly due to lack of agent-specific exposure information, lack of quantification of human exposure (absorbed dose), and lack of human dose-response information. Lack of data on these human data parameters is the reason for the high uncertainties associated with endocrine disruption at this time. Due to ethical and other considerations we will never have the level of data available in humans as are possible in laboratory animals.
potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	H-M, Availability of quantitative tissue concentration data and dose response information could have a very large impact on the degree of risk involved.
potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	(-)While recognition of the potential for ED effects is increasing, many substances with potential ED effects continue to be released into the environment and levels of population exposure may increase as background levels in the environment increase.
potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	M - Single events such as the unintentional PBB addition to animal feed in the 1970s can have significant ED consequences. However, for the most part ED emissions and releases tend to be low-level and chronic.
extent to which risks are currently reduced through in-place regulations and controls	The full nature and extent of ED risk is not yet known, while it is likely that some amount of underlying ED risk is controlled as a result of existing regulations aimed at other endpoints, few if any regulations address ED per se.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
large business/industry	H - There may be significant amounts of potential ED substances in consumer products. In addition, some ubiquitous substances (e.g., phthaltes) which may have ED effects are released with little or no control. In addition, pharmaceuticals and pharmaceutical breakdown products are not subject to complete environmental control.
small business industry	M - specialty chemicals
transportation	L
residential	H - improper/reckless use of pesticides, discharge of ED active compounds, pharmaceuticals, and over-the-counter medications in sewers, and improper disposal of toxics.

agriculture	H - Inappropriate application rates of pesticides on crops and/or inappropriate use of pesticides on certain crop types.
recreation	L
resource extraction	L
government	L
natural sources	L
contaminated sites	M - Contamination of soil and water
diffuse and non-NJ sources	
sediment	H - Contains elevated concentrations of many toxics (persistent organic pollutants)
soil	M - Contaminated sites may pose potential elevated risks
non-local air sources (including deposition)	M - Air deposition occurs across political boundaries - High for certain pollutants, e.g., mercury, TCDD
biota sinks	H/M -Soil/sediments, flora, and fauna

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
L-M, 2.5	M - 2.5	Y, 5	M - 2.5

FN: Hwg-ed.2/28

Issue: Extremely Low Frequency and Electromagnetic Fields (ELF/EMF)
Author: Debbie Wenke
Version: 08/09/00

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Stressor: **Extremely Low Frequency and Electromagnetic Fields (ELF/EMF)**

Rationale for why the CRP should not perform a full scale assessment on the potential impacts of nominal residential exposure to electromagnetic fields (EMF, magnetic and electric fields associated with the production, transmission, and use of electricity) on human health resulting from 60 Hertz electrical distribution lines (specifically excluded from this discussion are exposures which may occur in certain occupational activities and the use of household appliances and cellular radios).

Time-averaged residential exposures to EMF fields from electrical distribution lines in the United States (60-Hertz/second) are very weak compared to the earth□s background field and are usually much smaller than EMF intensities residents are exposed to from voluntary residential activities. That is, people with electric analog alarm clocks on a night stand, using kitchen mixers, microwave ovens, electric blankets, hair dryers, vacuum cleaners, electric shavers, electric tooth brushes, etc. have significantly higher exposures to EMF from these lifestyle activities than could reasonably be expected to occur from electrical distribution lines. Some epidemiology studies concerning exposure to residential EMF resulting from electrical distribution lines have shown some weak associations for increased rates of breast cancer, brain cancer, and leukemia. These findings have not been able to be duplicated using higher EMF exposures in laboratory animal models by the National Toxicology Program. In 1999, the National Institute of Environmental Health Sciences (NIEHS) concluded the scientific evidence that exposures to EMF pose a health risk is □weak,□ although the working group□s decision designated EMFs as a □possible human carcinogen.□ NIEHS gave advice that has been widely interpreted as an endorsement of a policy of □prudent avoidance,□ or taking simple and inexpensive steps to limit exposures. This same policy was put forward a decade ago by the Congressional Office of Technology Assessment and four years ago by the government of Sweden.

Human Health Issue Summary: Extremely Low Frequency/Electric and Magnetic Fields

What is it?
Electromagnetic fields (EMF) are produced by the generation, transmission, and use of electrical energy. U.S. standards for delivering electrical current place these fields in the extremely low frequency (ELF) range of 3 hertz (Hz) to 3,000 Hz. Magnetic fields exist adjacent to electric charges. Major sources of ELF magnetic fields are transmission and distribution lines, transformers, house wiring, appliances, train lines, and facilities that do electrogalvanizing, metal refining, induction heating, foundry work, and degaussing. Magnetic fields have been implicated in promotion of cancer, specifically childhood leukemia and chronic lymphocyte leukemia in adults.

What's at risk?
Statewide, nearly all of the population is exposed to ELF/EMF via overhead power lines. Electrical utility workers receiving greater exposure may be at increased risk for certain types of cancer. It is possible that children may be at a small, increased risk for certain types of cancers if their homes are near high voltage transmission lines or heavily-loaded distribution lines.

What are the human health impacts in New Jersey?
Studies to date have provided weak evidence connecting occupational exposure to magnetic fields (EMF) with adult chronic lymphocytic leukemia. Childhood exposure to magnetic fields might result in an additional 4-13 cases of leukemia statewide per year. However, the potential for any cancer from EMF is unclear and the number of attributable cancer may be zero.

Issue: Extremely Low Frequency and Electromagnetic Fields (ELF/EMF)
Author: Debbie Wenke
Version: 08/09/00

What's being done?
Guidelines exist to restrict ELF electric fields at the edge of transmission line rights-of-way to 3 kilovolts per meter (kV/m). Available evidence suggests that the effects of electric field exposure up to 20 kV/m are few and not harmful.

Issue: Formaldehyde
Author: Ken Jones, Michele Witten
Version: 01/17/02/

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Formaldehyde**
Description of stressor (including etiology)	Formaldehyde is the simplest aldehyde and is a chemical with many industrial and commercial uses, as well as a byproduct of combustion. The sources of combustion include petroleum in vehicles and boilers and from cigarette smoke. (Doull et al.) As with many pollutants, formaldehyde concentrations indoors can exceed outdoor concentrations. Formaldehyde can be released from fabrics, paints and pressed-wood products. Prior to 1980, Urea Formaldehyde Foam Insulation (UFFI) was a significant source of formaldehyde in some newly constructed buildings. For most indoor sources, the concentration released to the air decreases significantly over time. For this reason, homes with UFFI no longer have significant formaldehyde from that source (www.heimer.com)
Stressor-specific impacts considered including key impacts	Formaldehyde is an irritant affecting sensitive mucous membranes. One of the easily recognized symptoms of formaldehyde exposure is irritation of the eyes (Doull et al.). Formaldehyde affects lung function and raises the susceptibility towards infection. Lung function changes can be measured by observing expiratory volumes and respiratory resistance (Doull). Related to its affects on lung function, formaldehyde may be an important contributor to the onset of asthma. Formaldehyde is also considered an allergen (Dearman et al., 1997). Formaldehyde is considered a probable human carcinogen by the USEPA (USEPA, IRIS).
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate) Population(s)/ecosystem(s) exposed statewide	The irritant and lung function effects of formaldehyde are the result of inhalation. Formaldehyde is a significant contaminant of indoor environments, with very high levels recorded from 20-40 years ago when UFFI was a common building insulation material. Formaldehyde is a pervasive pollutant, therefore all New Jersey citizens are exposed. There are higher concentrations of formaldehyde modeled for urban areas as noted in the National Air Toxics Assessment (NATA) modeling results for the year 1996. (NATA)
Quantification of exposure levels statewide, including populations at significantly increased exposure	NATA modeling of formaldehyde concentrations provides a range of possible exposures for New Jersey counties. The highest reported concentration using this model is in Hudson County showing a median value of 4.1 ug/m3 (or about 3 ppb). Less urban counties such as Sussex and Atlantic show median values of less than 1 ug/m3 (or 0.8 ppb).

(include indoor air as separate category as appropriate)	Actual monitoring carried out in Camden offers supporting evidence for ambient concentrations possible in urban areas. In 1996, almost all of the approximately 100 readings showed detection of formaldehyde greater than 1 ppb and almost one third of the readings were greater than 10 ppb. (The NATA model suggests a statewide median concentration of about 1.5 ppb.) (NJ DEP). Studies outside of New Jersey provide some evidence of the prevalence of formaldehyde in indoor settings. One study in Louisiana, one in the Houston, Texas area and one in Austria all indicate that between 10 and 50 percent of homes may have formaldehyde concentrations greater than 0.1 ppm.
Specific population(s) at increased risk	Formaldehyde concentrations in outdoor air are greater in urban areas. The impacts in indoor air may also lead to risks to special populations. There are several studies identifying a range of concentrations in the workplace, schools and in homes. (Stock and Mendez, 1985; Lemus et al., 1998; Koeck et al., 1997) There does not appear to be a pattern in the distribution of increased formaldehyde in indoor environments, therefore it is hard to identify populations exposed to greater concentrations. However, asthmatics and those suffering from allergies may be more susceptible to formaldehyde's effects. Formaldehyde may be an important stressor to those suffering from multiple chemical sensitivities. This controversial health issue is based upon observations of specific populations that suffer physiological responses from concentrations of pollutants that show no impacts on the general population in clinical studies. One characteristic of exposures that is common in those suffering from Multiple Chemical Sensitivities is the presence of chemicals that surpass an odor threshold. (Kreutzeret et al.)
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations)	Based on NATA modeling, concentrations of formaldehyde in Hudson County are about 3-5 times that of less urban counties in New Jersey. The odor threshold for formaldehyde is approximately 300 ppb and there is evidence that indoor concentrations can reach that level in the presence of some consumer items. (EPA, www.epa.gov/iaq/formalde.html)
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	*The American Society of Heating and Refrigeration, and Air Conditioning Engineers has established a recommended limit of 0.1 ppm for formaldehyde in the home and workplace. Many studies use this threshold to identify impacts such as immune responses and episodes of asthmatic attacks. (Liu et al., 1991; Dearman et al., 1997)* *The cancer risk estimates in this assessment are based on EPA's unit inhalation cancer risk of $1.3x10^{-2}$ per mg (USEPA, IRIS).*
Risk Characterization Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile	For non-cancer effects, there are probably no outdoor exposures that approach levels known to result in impacts. However, some studies show impacts to asthmatics and allergic responses at levels of formaldehyde that are regularly found in homes, particularly those homes that are recently built, remodeled or painted. Exposure to median concentrations of formaldehyde in New Jersey (1.5 $\mu g/m^3$) may lead to 23 excess cases per million population. This translates to about 3 additional cancer cases per year for the New Jersey population. For Hudson County, the county with the highest estimated exposure, less than one additional cancer per year from ambient formaldehyde exposure

population risk, etc.)	would be expected.
Risk estimate(s) by population at risk (Children)	There are no studies estimating proportions of children that may be susceptible to the increased risk of infection. The studies implying the connection simply note that there may be statistical increases in infections among those children exposed to formaldehyde (Leikauf).
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	M - Formaldehyde exhibits two sets of health effects. Cancer is possible from exposure to ambient concentrations and is related to the existence of low concentrations over long periods of time. Most acute reactions to indoor exposure are reversible and the symptoms largely disappear when the patient is moved to fresh air. However, some researchers do conclude that continuous exposure to elevated formaldehyde in the home may lead to chronic effects including allergic sensitization.
Size of population(s) affected	The entire New Jersey population is exposed to concentrations that may increase their incidence of cancer.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	M – Most of the risk calculations in this report are based on the carcinogenicity of formaldehyde. Formaldehyde is classified as a "probable" human carcinogen based on some human evidence but mostly animal studies. The calculation of the risk slope factor is subject to a wide range of variability. As with many studies of chemical carcinogenesis, the extrapolation of animal data to humans may lead to significant uncertainty regarding the actual carcinogenic impact on humans. In particular, questions have been raised about the relevance of the nasal passage tumors produced in rats for human exposure and risk.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	L/M – There are no greater number of studies focusing on formaldehyde than other environmental pollutants, so it is difficult to speculate that future findings will result in a significant change in the risk estimate. The decreased use of Urea Formaldehyde Foam Insulation has taken some public focus away from formaldehyde.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =, where + is improvement)	0 - We do not know of any immediate strategy to reduce formaldehyde emissions other than general strategies to control combustion processes. Therefore, formaldehyde emissions are not expected to show dramatic changes in the near future. Historical review of formaldehyde emissions and ambient concentrations have not shown large trends. (NJ DEP)
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L - We do not believe that formaldehyde releases from accidental events will generate significantly elevated risks.
Extent to which risks are currently reduced through in-place regulations and controls	The removal of Urea Formaldehyde Foam Insulation from the decreased incidence of acute indoor exposure. There remain many products that emit formaldehyde and there is no additional regulation for these products.
Relative Contributions of Sources to Risk (H,M,L)	The EPA's Cumulative Exposure Project estimates that greater than 95% of formaldehyde releases come from mobile sources with the remaining 5% derived from area and point sources. (US EPA, (CEP))
Allocation of stressor-specific risk to primary NJ sources	
	L

Large business/industry	
Small business industry	L
Transportation	H - Outdoor
Residential	H – Indoor
Agriculture	L
Recreation	M (off road vehicles and boats)
Resource extraction	L
Government	L
Natural sources	L
Contaminated sites	L
Diffuse and non-NJ sources	There are background sources of formaldehyde that may contribute as much as 20% as much as mobile sources.
Sediment	L
Soil	L
Non-local air sources (including deposition)	L/M
Biota sinks	L

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)

Cancer 3-M	3-M	3-M	3-M
Indoor Exposures 4-M/H	4-M/H	3-M	4- M/H
			4-M/H

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Issue: Formaldehyde
Author: Ken Jones, Michele Witten
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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Greenhouse Gases**
description of stressor (including etiology)	Most scientists believe that the increase in emissions of "greenhouse gases" (i.e., carbon dioxide, nitrogen oxide, methane, and chlorofluorocarbons) due to human activity has gradually caused a rise in the average temperature of the planet. Most of the human activity causing these emissions come from auto usage, power plants, and other industrial sources (EPA 1997).
stressor-specific impacts considered including key impacts	Since the impacts of global warming are still relatively uncertain and long term in nature, we can only look toward expert predictions. There are five major concerns about the impact of global warming to human health (Patz & McGeehin 1998): increase in heat strokes and heat related deaths due to hotter summers; increase in respiratory diseases due to an increase in air pollution; increase in deaths from violent storm and flood activity; decrease in water quality due to unclean storm water or algae blooms; leading to sickness related to poor water or food (e.g., fisheries) quality; and increase in diseases carried by insects (e.g. malaria, Lyme disease).
Exposure Assessment	
exposure routes and pathways considered (include indoor air as appropriate)	As mentioned above, exposure routes and pathways are mostly from outdoor air, water, disease carrying insects, and possibly food sources.
population(s)/ecosystem(s) exposed statewide	The total state population is exposed; however, people near flood zones and coastal areas will be more susceptible to deaths from violent storm and flood activity. Also, people located in areas of the state that currently have high levels of ground ozone might be increasingly exposed to respiratory diseases.
quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	This would equate to the total state population. The 2000 US Census estimate of the state population is 8,414,350.

specific population(s) at increased risk	While the total population of the state is at risk, the following segments of the population might be particularly exposed: elderly, infants, and people with cardiovascular or respiratory diseases people living in flood zones or coastal communities
quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	Do not have quantifiable numbers for these sub-populations.
Dose/Impact-Response Assessment	
quantitative dose/impact-assessment employed for each population considered	None available. A qualitative association is assumed.
Risk Characterization	
risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	No quantitative risk assessment was performed.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	There are five main categories of risk: Heat Related Impact A rise in temperatures will lead to more intense summer heat waves. Hot weather stresses our bodies due to the increased demand on our cardiovascular system for physiological cooling and, therefore, could cause an increase in deaths and heat related illnesses (heat strokes, heat exhaustion, heat cramps, fainting, and heat rash). Heat also aggravates existing medical problems. The elderly, infants, and people with cardiovascular or respiratory diseases would be the most susceptible. NJ can be particularly affected by this impact because of the irregular amount of heat waves we currently have (EPA 1997). People who live in hot climates have adapted their lifestyles to cope with excessive heat but this is not necessarily the case for people in temperate climates such as NJ. One study suggested that a growth in heat waves could increase by five times the number of heat related deaths in Newark from the current level of 25 deaths to 125 (although the study might not have reflected the use of air conditioning correctly) (EPA 1997). Because poorer families might lack air conditioners, they might be particularly susceptible.

	An increase in water borne diseases will likely cause an increase in illnesses in NJ. We could curtail this impact by improving our water treatment facilities. Although harmful algal blooms are a complex result of many factors, and do not necessarily result from elevated temperatures, higher surface temperatures in the ocean could contribute to the increased intensity, duration, and extent of harmful algal blooms (EPA 1997 & Shriner & Street 1998). These blooms can damage habitat and shellfish nurseries, can be toxic to humans, and can carry bacteria like those causing cholera. Brown algal tides and toxic algal blooms already are prevalent in the Atlantic Ocean. Warmer ocean waters could increase their occurrence and persistence. NJ is susceptible because of its active fishing and restaurant industry along the shorelines of the Atlantic Ocean. Diseases Spread by Insects Warmer climates could expand the habitat of disease carrying insects. These insects (e.g., mosquitoes) might move north and increase the spread of diseases such as malaria, dengue fever, and encephalitis. We would also need to be concerned about an increase in ticks carrying Lyme disease. Ticks are most active in hot and humid climates. Since mosquitoes already inhabit many areas in NJ, our state could be adversely affected. Scientists believe an outbreak in NJ that occurred in the 1990s was aided by exceptionally hot and humid weather, which reduced the development time of the malaria parasite making northern mosquitoes infectious. One study estimated an increase in global temperatures could result in an increase in the vectorial (disease carrying) capacity of mosquitoes 100 times in temperate countries such as the US (Shriner & Street 1998). Because of our high living standards and health care infrastructure both in NJ and the US, we would expect that we would not suffer any major outbreaks; however, even a small increase in highly infectious diseases might financially strain our current system (Bernard 2001 & Shriner & Street 1998). Our extensive use of pesticides might also help curb major outbreaks although the use of pesticides could also have adverse health impacts depending on how they are used. Other Concerns There are some other general changes that might occur that could have indirect human health impacts in NJ. Because of increased ozone we could suffer a reduction in agricultural crop yields. Changing weather patterns could jeopardize access to traditional foods obtained from land and water. Because of these adverse impacts on our food supply, we could see a rise in diet related problems leading to obesity, cardiovascular disorders, and diabetes. Also, one of the major concerns about global warming is the complexity of the issue. A change in climate can lead to unforeseeable changes to our current land uses. These changes will probably occur gradually but can have enormous consequences. Global warming can particularly effect NJ due to our large population dependent on our coastal lands. However, any impacts to the health of NJ's population will be very long term in nature.
	Some outcomes might reduce some of the heat related impacts. Deaths due to cold weather should decrease; however, experts believe that this will only partially offset the increases in morbidity and mortality from heat waves (Shriner & Street 1998). Also, people should eventually become acclimatized to the warmer weather. This should further reduce heat related morbidity and mortality; however, this change in people's behavior will probably be gradual over many years. Eventually, air conditioning might

	become more prevalent in NJ. Also, warning systems such as one in Philadelphia might be used to alert the public to extreme hot weather systems (Shriner & Street 1998). Air Pollution Impact Global warming could cause a rise in ground level ozone. High temperatures, strong sunlight and stable air masses will tend to increase ground level ozone. Increased air conditioner usage will also cause a rise in natural hydrocarbon emissions from power plants. High levels of ozone cause damage to lung tissue and reduce pulmonary function and sensitize airways to other irritants and allergens. People with respiratory diseases such as asthma, people with allergies, the elderly, and children will be impacted the most. However, healthy people will be affected as well. At elevated levels of ground level ozone, normally healthy people can experience chest pain, coughing, nausea, and pulmonary congestion. Again, NJ might be particularly susceptible to this impact due to the high level of auto activity combined with hot weather, especially the northeastern section of the state. Current ozone concentrations exceed the national health standards for ozone throughout the state and most of the state is classified as an "extreme and severe" non-attainment area for ozone. The EPA has estimated that a 4°F warming of New York City, with no other changes in weather or emissions, could increase concentrations of ozone by 4% (EPA 1997). NJ would probably experience a similar rise in ozone. Partially offsetting these ozone increases could be continued successes in decreasing auto and power plant emissions. Violent Storm Activity Experts expect an increase in violent storms such as hurricanes and tornadoes with global warming. This increase in storms is due to an increase in convective activity. While tornadoes are not a common occurrence in NJ, hurricanes and floods occasionally hit NJ and can cause loss of life and injury. Hurricane Floyd from 1999 can provide an example of what could become a more frequent occurrence. Flooding leads not only to drowning deaths but also can lead to destruction of food supplies and outbreaks of disease as a result of a breakdown in sanitation services. It can cause a release of dangerous chemicals from storage sites and waste disposal sites into floodwaters. Storm runoff can wash chemicals from agricultural lands and industrial sites into water supplies (Shriner & Street). Water Quality Impact The EPA believes that poor sanitation, poor erosion control management, coastal sewage release, and contamination of drinking water from agricultural fertilizers and hazardous waste may all increase the likelihood of water-borne disease. Drinking water supplies can be subject to saltwater intrusion from higher ocean levels caused by the melting polar ice caps and from thermal expansion of oceans that will result from global warming. States along the East Coast such as NJ might be particularly hard hit (Patz and McGeehin 1998).
size of population(s) affected	The total population of the state would be affected; however, the following segments of the population might be particularly exposed: elderly, infants, and people with cardiovascular or respiratory diseases; and

	people living in flood zones or coastal communities. We do not have population levels for the sub-population groups although the total population of New Jersey based on the 2000 census was 8,414,350.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	H: While most scientists agree that the earth is warming, there is still a great deal of uncertainty related to the extent and impact of global warming. The long-term nature of the impacts adds to this uncertainty.
potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	H: As mentioned earlier, there is a great degree of uncertainty related to the impact of global warming on the earth in general. It is even more difficult to try to develop a risk estimate on a small sub-area of the globe such as New Jersey. There is still a variety of differing opinions among experts on the impact of global warming.
potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, -, =, □ where + is improvement)	-- Most scientists believe that global warming will occur and gradually get worse. Even if the world can agree on a treaty to decrease greenhouse gases, most scientists believe that a reduction in emissions will not be enough to stop the increase in the greenhouse effect and produce warmer temperatures in the coming decades. A treaty on greenhouse gas emissions would only reduce the extent of the warming trend. Adverse human health impacts associated with this trend are likely in NJ but they can be mitigated if this wealthy state's residents decide to do so.
potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	H: One of the main impacts of global warming could be from the detrimental results from increasing violent storm or flooding activity.
extent to which risks are currently reduced through in-place regulations and controls	M: Heat related impacts could be offset by warning systems alerting people to extreme hot weather such as the one in place in Philadelphia. Other extreme weather warning systems (e.g., hurricane warnings) should ease the impact of violent storms. Controls on building in flood zone areas should limit the number of people impacted by flooding events. Regulations on some greenhouse gases such as Nitrous Oxides and emissions regulations for vehicles have helped to slow down the growth in ground level ozone. Regulations concerning water treatment should help curb detrimental impacts from decreases in our water quality. Our current health care infrastructure should help curtail any major disease outbreaks.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
large business/industry	H
small business industry	H
Transportation	H

Residential	H
Agriculture	M
Recreation	L
resource extraction	M
Government	M
natural sources	M
Contaminated sites	L
diffuse and non-NJ sources	
Sediment	L
Soil	L
non-local air sources (including deposition)	H
biota sinks	L

Human Health Issue Summary: Greenhouse Gases

What is it?
Increasing amounts of Greenhouse Gases in the atmosphere cause a gradual rise in average global temperatures, referred to as global climate change. More than 80% of the gases are the result of the combustion of fossil fuels and atmospheric concentrations of carbon dioxide have increased nearly 30% since pre-industrial times. The buildup of heat- trapping gases in the atmosphere is linked to a gradual rise in sea level and an increase in intense storm activity.

What's at risk?
There are five human health concerns associated with the global warming trend: increases in heat-related deaths, increases in respiratory diseases due to increased air pollution, increases in storm and flood-related deaths, increases in water-borne illnesses, and increases in diseases carried by insects. Should the hypothesized effects of climate change materialize, all of New Jersey's population would be susceptible to health problems related to an increase in heat waves and air pollution. The elderly, infants, and people with cardiovascular or respiratory diseases would be particularly vulnerable. People living in coastal areas would be at a greater risk from the effects of violent storms and flooding. It is impossible to predict the extent of secondary effects related to increases in disease caused by poor water quality or by the northern migration of disease-carrying insects.

What's being done?
A treaty on greenhouse gas emissions may result in a slower warming trend, but most scientists agree that reducing emissions will not be enough to stop the increase in the

greenhouse effect that will produce warmer temperatures in the coming decades. Impacts can be managed to some extent. Flood damage can be limited by controlling development in flood zones. New Jersey's existing health care system will to some extent be able to contain any major disease outbreaks.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
L/1	H/4	Y/3	L/2
			L

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Issue: Hantavirus
Author: Tom Atherholt
Version: 05/02/01

New Jersey Comparitive Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Hantavirus**
Description of stressor (including etiology)	Hantavirus is an airborne viral pathogen contracted via inhalation of aerosols generated from disturbed rodent saliva or excreta (CDC). Other viruses may have a similar mode of transmission (see note). Note: The Centers For Disease Control and Prevention reported on 3 fatalities in California which may have been the result of viral infection following contact with rodent feces (Byrd *et al.*, 2000). Viral RNA very similar to Whitewater Arroyo (WWA) virus was found in specimens from each of these patients. Some of the symptoms of these infections are similar to that following hantavirus infection (see below). WWA is a member of the arenavirus family. In Africa and South America, some arenaviruses are capable of causing serious, often fatal diseases such as Lassa fever and lymphocytic choriomeningitis. WWA is found in North America among woodrats (*Neotoma* spp.). Although the abundance and habits of woodrats suggest that potential contact between *Neotoma* spp. and humans is limited, nine of the 20 known species of *Neotoma* inhabit North America and at least five of these species may harbor WWA. The geographic range of these species incorporates most of the U.S.
Stressor-specific impacts considered including key impacts	Severe, often fatal pulmonary illness.
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	Inhalation of aerosols generated following the physical disturbance of rodent saliva or excreta in closed-up spaces. Other possible exposure routes include direct contact of the contaminated rodent saliva/excreta with broken skin (*e.g.*, cuts), exposure to the eyes, or possibly contaminated food or water. Persons have become infected after being bitten by infected rodents (CDCa). There is no known insect transmission, no known person-to-person transmission nor any known nosocomial (hospital-acquired) transmission (CDCa).
Population(s)/ecosystem(s) exposed statewide	Anyone conducting activities (*e.g.*, cleaning) which result in the generation of dusts or aerosols in indoor structures containing high concentrations (nests) of deer mice (*Peromyscus maniculatus*) or, for the northeastern US, white footed mice (*P. leucopus*).

Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	No data.
Specific population(s) at increased risk	In addition to the general exposure scenario (above), several occupational groups may be at increased risk. These include grain farmers, field biologists, and agricultural, mill, construction, utility and feedlot workers (CDCa).
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	No data.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	None.
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	Risk is considered extremely low. Since hantavirus and hantavirus pulmonary syndrome ("HPS") were first characterized in 1993, until May 28, 1999, CDC has confirmed only 217 cases of HPS in the United States. Most cases have occurred in the southwestern US. There has not been any documented case in NJ (or any in any northeast state with three exceptions. There were 2 confirmed cases in NY, two in PA, and one in RI) (Leslie *et al.,* 1999). Note: The hantaviruses in the northeast US appear to be distinct from that in the southwest US and have been given strain designations of "New York-1" and "Monongahela". The NY-1 virus preferentially infects the white-footed mouse (*Peromyscus leucopus*) while Monongahela infects both *P. leucopus* and the deer mouse (*P. maniculatus*) which is the host for the southwest US strains (CDCa; Monroe *et al.*, 1999; Rhodes *et al*,. 2000).
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	HPS includes headache, fever, and myalgia, followed by pulmonary edema, often leading to severe impairment of lung function. HPS has a case-fatality ratio of 43% making HPS a rare, but serious disease. There is no known treatment other than supportive care. (Leslie *et al.*, 1999).
Size of population(s) affected	No documented cases in NJ.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	L.

Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	L.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, -, =, □ where + is improvement)	0
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L.
Extent to which risks are currently reduced through in-place regulations and controls	None.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	Low. Rodent infested structures.
Small business industry	Low. Rodent infested structures.
Transportation	None.
Residential	Low. Rodent infested homes or other structures.
Agriculture	Low. Rodent infested barns or other structures.
Recreation	Low. Rodent infested buildings at campgrounds and the like.
Resource extraction	None.
Government	None.
Natural sources	All cases arise from natural sources.
Contaminated sites	None.
Diffuse and non-NJ sources	

Sediment	None.
Soil	None.
Non-local air sources (including deposition)	None.
Biota sinks	Rodents are the source of all hantavirus and Whitewater Arroyo virus (see note in first section).

Issue: Hantavirus
Author: Tom Atherholt
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Human Health Issue Summary: Hantavirus

What is it?
Hantavirus is an airborne viral pathogen generated from disturbed rodent saliva or droppings. It can be contracted by humans via inhalation of contaminated aerosols, or possibly through contact with broken skin or rodent bites. Once contracted, the infection may lead to pulmonary illness, which is often fatal.

What's at risk?
Anyone conducting activities (e.g., cleaning) which result in the generation of dusts or aerosols in indoor structures containing large numbers of deer or white-footed mouse nests. People who are occupationally exposed—grain farmers, field biologists, mill, construction, utility, and feedlot workers for example—may be at increased risk.

What is are the human health impacts in New Jersey?
Risk is considered extremely low. There have been no known cases of hantavirus in New Jersey and a little over 200 cases in the U.S. since the disease was first characterized in 1993. In the northeastern U.S., there have been 2 confirmed cases in New York, 2 in Pennsylvania, and 1 in Rhode Island.

What's being done?
There are no regulations or controls in place. Hantavirus is a rare, but serious disease with no known treatment, other than supportive care.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
L or 1	L or 1	Y (not definitively established)	L or 1
			L or 1

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Byrd, R.D. et al.. 2000. Fatal illnesses associated with a New World arenavirus - California, 1999-2000. Morbidity and Mortality Weekly Report 49(31): 709-711, August 11.

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Issue: Hantavirus
Author: Tom Atherholt
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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Indoor Asthma Inducers**
Description of stressor (including etiology)	Those chemicals and particulates, both biological and non-biological that induce asthma in the indoor environment including: Cockroach feces/antigen dust mites animal/pet dander Rodent protein (found in urine) Environmental tobacco smoke[1]
Stressor-specific impacts considered including key impacts	The key impact of exposure to these indoor allergens considered in this report is asthma. Asthma is a condition which affects the small airways of the lungs. Asthma is an allergic reaction to exposures in the environment. People with asthma have sensitive or "twitchy" airways. When these sensitive airways are exposed to certain triggers- (i.e., biological and non-biological stressors), the airways can narrow, leading to difficulty in breathing. The narrowing of the airways is due to (1) tightening of the muscles around the airways and (2) the production of excess mucus in response to inflammation of the airways. The result is a reduction of airflow in and out of the lungs. Therefore, impacts considered include asthma induction and exacerbation.
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	Inhalation of the allergens into the lungs is the mechanism that is required to produce the immune response that leads to asthma induction and exacerbation.
Population(s)/ecosystem(s) exposed statewide	All populations and ecosystems statewide are exposed to these triggers, as they are ubiquitous in the environment.
Quantification of exposure levels statewide (include indoor air as separate category as appropriate)	Quantification of exposure levels does not exist at the present time on a statewide level, yet, since most of the identified stressors are common place, exposures to these stressors would be widespread.
Specific population(s) at increased risk	Animal handlers, veterinary workers (vets and technicians), livestock workers, garment workers, horse handlers workers, asthmatics, children and adults in low-income communities are at increased risk for exposure and are more

	susceptible to the morbidity and mortality associated with exposure. Populations with increased risk also vary by gender, and race. According the American Lung Association, the age adjusted asthma mortality rate for asthma from all causes in white males has increased from 0.8 to 1.0 per 100,000 between 1979 and 1997, an increase of 25%. During the same period, the mortality rate in white females increased by 63%. The age adjusted mortality rates increased by 68.4% in black males and 95% in black females over this time span. Mortality rates in non-white (all races other than white) males and females exhibited increases of 66.7 percent over this time[2].
Quantification of exposure levels to population(s) at increased risk (due to factors other than exposure) (include indoor air as separate category as appropriate)	Individuals with atopic disease (i.e., those with an inherited tendency to develop asthma, hay fever, and other IgE mediated immune responses) are more likely to develop asthma upon exposure to the listed stressors as well as stressors that do not cause an allergic response in normal non-atopic individuals. Furthermore, individuals in low-income communities may be at increased risk for mortality associated with asthma because of inadequate access to medical care. Various studies conducted in urban areas show much higher asthma mortality associated with low income and minority populations[3].
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	These pollutants trigger their affects at □g and ng quantities in asthmatics. For those with atopic disease, the quantities required for disease to occur may be smaller and may not be limited to the stressors listed in the hazard identification list above.
Risk Characterization Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	1994-1996 Data Age Adjusted Death Rate (by 1,000,000)[4] Total 12.9 White 9.4 Black 35.0 Hispanic N/A

Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Hospital Admission Rates 1996 Baseline Data (by 100,000) Total 203 White 132 Black 472 Hispanic 292 Hospital Admission Rates for Children <5 yrs of age All Children <5 610 White non-Hispan. 344 Black non-Hispan. 1,512 Hispanic 684 Additionally, data from the American Lung Association, estimates that in the year 2000, there will be 316,307 episodes of adult asthma, and 122,804 episodes of pediatric asthma in New Jersey *. These figures differ from the numbers estimated for 1999 with a decrease in the number of predicted pediatric cases (-16.5) and an increase in the predicted numbers for adult asthma (+5.8). It must be noted that these numbers are estimates and are not derived from a complete census or case registry of disease. Furthermore, data is self reported and at the present time, no data exists on a statewide basis for asthma incidence or prevelance[5]. *(These estimates are based on those conditions that prompt at least one doctor visit or one day of restricted activity). Asthma attacks can range in severity from mild to moderate to severe attacks requiring hospitalization and in some cases an attack can lead to death. Mortality Asthma was listed as the underlying cause of death in 5,667 of 2.3 million deaths in the US during 1996. Asthma mortality primarily affects adults, with approximately 67% occurring at or after 45 years of age. Males tend to have higher rates of asthma than females until about age 25, after which females have higher rates for the rest of their life span. In contrast, the morbidity burden is much greater.
	Morbidity

	Asthma rates are highest in the young, less than 5 years of age, with a second, lower peak in the group age 65 or more. In the young, hospitalization rates are higher for males, whereas among adults, females have higher rates. Hospitalization rates are elevated in urban areas with high levels of poverty and/or minority populations. Readmission's can account for up to approximately 20% of hospitalizations with readmission rates being higher in the younger age groups. And, hospitalization rates are highest on the east coast, but this trend is apparent not only for asthma, but also for all causes of hospitalizations. Furthermore, outpatient care in the form of Emergency Room visits have been tracked in the US only since 1992. Similar to hospitalizations, African Americans and women have slightly higher rates[6].
Size of population(s) affected	The sizes of population affected are hard to determine as asthma registries do not exist and hospitalization rates/ER or outpatient visits may not account for all occurrences of asthma. However, as noted above there will be an estimated 316,307 cases of adult asthma and 122,804 cases of pediatric asthma in New Jersey in the year 2000.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	High The exact nature and contribution of each of the stressors to the induction and exacerbation of asthma is dynamic and as asthma is a highly complex disease associated with familial, infectious, allergic, socioeconomic, psychosocial and environmental factors,[7] the uncertainties in assessing risk are very high. Furthermore, triggers and pollutants in the outdoor environment as well as allergies to food and stress play a role in asthma induction and exacerbation and therefore, not all asthma can be attributable to indoor air pollution.
Potential for significant future change in this risk estimate (H,M,L) and brief description	High As noted above, as the etiology of asthma is better understood, risk estimates could change. However, the number of cases continues to grow.
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	Low
Extent to which risks are currently reduced through in-place regulations and controls	Low
Relative Contributions of Sources to Risk (H,M,L)	Not yet known

Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	N/A
Small business industry	N/A
Transportation	N/A
Residential	High
Agriculture	N/A
Recreation/ Outdoor * *Outdoor added to original template*	High
Resource extraction	N/A
Government	N/A
Natural sources	N/A
Contaminated sites	N/A
Diffuse and non-NJ sources	
Sediment	N/A
Soil	N/A
Non-local air sources (including deposition)	N/A
Biota sinks	N/A

[1] These allergens are not listed in any specific order of importance/ allergenicity.

[2] *American Lung Association, Trends in Asthma Morbidity and Mortality*

[3] Clearing the Air.

[4] Healthy New Jersey 2010 Draft Data.

[5] *Estimated Prevalence and Incidence of Chronic and Acute Lung Disease by Lung Association Territories, 2000.*

[6] Clearing the Air.

[7] Legislative Commission on Toxic Substances and Hazardous Wastes, Childhood Asthma and Environmental Risk Factors, Children at Risk, Spring 2000 Issue Paper.

Human Health Issue Summary: Indoor Asthma Inducers

What is it?
Asthma is a complex condition affecting the small airways of the lungs. An initial exposure to allergens, viruses, pollution, or certain chemicals may induce the inflammation that leads to asthma symptoms in some individuals. Indoor asthma inducers include dust mites, animal/pet dander, mold, rodent protein, cockroach feces, and tobacco smoke. Asthma episodes may include lung inflammation, difficulty breathing, or in some cases, death. Episodes are caused by inhalation of these same inducers, or other asthma triggers that may occur in either the indoor or outdoor environment, once an individual develops asthma.

What's at risk?
The risk is statewide, with certain occupational groups at higher risk, such as veterinarians or livestock workers. Children and adults in low-income communities are at increased risk, for reasons that are not entirely clear. African Americans are three to four times more likely than Caucasians to be hospitalized for asthma, and four to six times more likely to die from asthma. Individuals with atopic disease, an inherited tendency to get asthma, are more likely to develop asthma when exposed to these inducers. Estimates indicate that one third to one half of the U.S. population may be atopic.

What are the human health impacts in New Jersey?
Hospitalization and outpatient visits may not include all episodes, but about 316,000 episodes of adult asthma and 123,000 episodes of asthma in children are estimated to occur in New Jersey in a given year (based on 2000 estimates). However, not all asthma can be attributed to indoor air pollution.

What's being done?
Currently there are few controls placed on indoor air quality, with the exception of restrictions on smoking in some public areas.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
4 - H	4 - H/M	Y - 5 (minority and low income communities)	4 H/M

References:

1. Allergy and Asthma Specialists.

2. American Lung Association, *Trends in Asthma Morbidity and Mortality*, http://www.lungusa.org/data/

3 Asthma Victoria, *About Asthma.*

4. American Lung Association, *Estimated Incidence and Prevalence of Chronic and Acute Lung Disease by Lung Association Territories 2000*, http://www.lungusa.org.

5. Healthy New Jersey 2010 Draft, Chapter 4-Preventing and Reducing Major Diseases.

6. Legislative Commission on Toxic Substances and Hazardous Wastes, *Childhood Asthma and Environmental Risk Factors, Children at Risk,* Spring 2000 Issue Paper.

7. USEPA, *Indoor Air Pollution, An Introduction for Health Professionals*, US Government Printing Office Publication No. 1994-523-217/8132.

8. Institute of Medicine, Committee on the Assessment of Asthma and Indoor Air. Clearing the Air: Asthma and Indoor Air Exposures. Division of Health Promotion and Disease Prevention, 2000.

Issue: Indoor Microbial Air Pollution
Author: Tom Atherholt
Version: 03/15/00

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Indoor Microbial Air Pollution**
Description of stressor (including etiology)	Excessive growth of normally free-living molds, mainly species of *Penicillium* spp. and *Stachybotrys atra (*a/k/a *chartarum)*, less often *Aspergillus* spp*., Paecilomyces, Fusarium*, in presence of warm temperatures and water-saturated or high moisture-impacted organic materials such as lumber, dry wall, ceiling tiles, furniture, carpet backing, wallpaper, books, papers, or cellulose-based insulation material, straw or hay. May also grow in air heating/cooling ducts and/or filters or in humidifiers using air-derived dust or dirt as the growth medium. Low (outdoor) levels of airborne spores usually do not cause any harm, except where outdoor levels become elevated due to nearby compost facilities or similar sources of spores (see separate assessment for outdoor air pollution).
Stressor-specific impacts considered including key impacts	For people with normal immune systems, infection without illness or else mild to severe adverse health effects in persons of all ages. Adverse health effects include skin irritation and rashes, eye, nose and throat irritation, wheezing, headaches, dizziness, fatigue, asthma, and diarrhea. For persons with asthma, allergies, diabetes mellitus, or weakened immune systems, adverse affects may be more severe. Such individuals are at increased risk for fever, shortness of breath, seizures, lung infections such as aspergillosis, lung damage, memory loss, and neurological damage. Infants less than 6 months of age are at increased risk for pulmonary hemorrhage or hemosiderosis (dark discoloration of the lung) with chronic cough, congestion, anemia, occasionally resulting in death (17% mortality rate for infants exposed to elevated levels of *Stachybotrys* spores with pulmonary hemorrhage in Cleveland between 1993-1996)(1).
Exposure Assessment	
Exposure routes and pathways considered **(include indoor air as appropriate)**	Inhalation of or dermal contact with toxin-containing spores of certain molds (see above) growing inside buildings that contain: standing/stagnant water in air conditioning systems; excessive moisture levels due to high humidity (^{3} 70 %) or from malfunctioning clothes dryer vents; saturated cellulose-based materials (see above) due to flooding, leaking roofs of plumbing fixtures; stored hay or leaves such as in barns; and other areas with potentially high mold levels such as antique shops, greenhouses, saunas, mills, construction areas, flower shops, summer homes (1).
Population(s)/ecosystem(s) exposed statewide	Population exposed to unsafe concentrations statewide is unknown: the population is probably in the hundreds if not more of NJ's 8 million residents. The amount of exposure which occurs in private residences vs. that which occurs

	in public and commercial buildings is not known.
Quantification of exposure levels statewide, including populations at significantly increased exposure **(include indoor air as separate category as appropriate)**	Under suitable environmental conditions, anyone can be potentially exposed. Populations at significantly increased exposure include some farmers, antique shop workers, greenhouse/flower shop workers, and anyone occupying a structure with excessive mold spore levels as described above. Owners of some summer homes in high moisture areas such as the coast may be subject to higher exposure levels. No quantitative data available. Levels are significantly higher than outdoor levels.
specific population(s) at increased risk	Statewide population of persons at increased risk, such as those with asthma, allergies, diabetes mellitus, persons with weakened immune systems and infants less than 6 months old, that are exposed to unsafe levels of mold spores in indoor air, is not known.
quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) **(include indoor air as separate category as appropriate)**	No quantitative data available.
Dose/Impact-Response Assessment	
quantitative dose/impact-assessment employed for each population considered	Unknown. Airborne spore concentrations that cause no effect in some people may cause mild or severe adverse effects in other people. Interindividual differences in susceptibility are common and can be quite large.
Risk Characterization	
risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	
assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Frequency of severe symptoms (e.g., pulmonary hemorrhage, neurological effects, and death) is rare. Frequency for milder symptoms is probably also low, but is likely to be higher than for severe symptomology.
size of population(s) affected	
assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	(assessment not possible)
potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	L

potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, -, =,º where + is improvement)	0
potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	N/A
extent to which risks are currently reduced through in-place regulations and controls	No regulations.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
large business/industry	L
small business industry	L
transportation	L
residential	M
agriculture	L
recreation	N/A
resource extraction	none
government	L
natural sources	H (spore source is soil/vegetation)
contaminated sites	N/A
diffuse and non-NJ sources	
sediment	none
soil	H (spore source is soil/vegetation)
non-local air sources (including deposition)	N/A
biota sinks	none

Issue: Indoor Microbial Air Pollution
Author: Tom Atherholt
Version: 03/15/00

Human Health Issue Summary: Indoor Microbial Air Pollution

What is it?
Indoor microbial air pollution is caused by excessive growth of bacteria, fungi, or algae in warm, wet materials including lumber, ceiling tiles, books and papers, insulation, or hay. Microbes may also grow in central air systems and filters, or in humidifiers. Indoor microbial pollution plays a role in "sick building syndrome," a group of symptoms associated with poor indoor air quality, particularly in airtight buildings with central air systems. Health effects from airborne microbial pathogens include respiratory infection, ranging from flu-like, or pneumonia-like symptoms to possible neurologic damage, pulmonary hemorrhage, and even death. Fungal toxins may cause flu-like symptoms, dermatitis, or diarrhea, as well as possible tissue and organ damage. A range of allergic responses, including asthma, are also possible (see separate report on Indoor Asthma Inducers).

What's at risk?
Airborne spores that cause no effect in some people may cause mild to severe effects in others. Persons with asthma, allergies, or weakened immune systems, and infants less than 6 months old are at increased risk, and may show more extreme reactions. At higher occupational risk are farmers, antique shop workers, greenhouse workers, or anyone occupying areas with excessive mold, or high moisture. Office workers in airtight buildings may be at risk for developing symptoms of sick building syndrome.

What are the human health impacts in New Jersey?
The population exposed to unsafe concentrations statewide is unknown, but it is estimated that hundreds of people are affected by indoor microbial air pollution each year. Incidence of severe symptoms, such as pulmonary hemorrhage, neurological effects, or death, is rare.

What's being done?
Overall, indoor air pollution is increasing, but there are no regulations or standards for maintaining indoor air quality.

Severity of specified health effects at current levels of exposure (H,M,L)	**Size of population at significant risk for each health effect (H,M,L)**	**Are there discreter communities at elevated risk? (Y,N)**	**Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L)**
L	L	Yes	L

1. Centers for Disease Control and Prevention: www.cdc.gov/nceh/pubcatns/facts/molds/molds.htm. Envirovillage: www.envirovillage.com/papers/N0000100001.htm. CBS News: cbsnews.cbs.com/now/story/0,1597,167321-412,00.shtml. Case Western Reserve University: gcrc.cwru.edu/stachy/default.htm.

Issue: Lead
Author: Mark Maddaloni
Version: 01/02/01

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework **Findings**

Hazard Identification	
Stressor	**Lead** Lead (Pb), including inorganic divalent salts (e.g., lead oxide, lead sulfide, and lead chloride) and organic compounds (e.g., lead carbonate, tetraethyl lead, and lead acetate)
Description of Stressor (including etiology)	Lead is a naturally occurring bluish-grey element (metal) found in small amounts[1] (.002%) in the earth's crust. In 1996, Missouri and Alaska accounted for 93% of the total (436,000 metric tons) lead mine production. New Jersey is a minor producer of lead, but 46 facilities[2] report the processing of lead on site. In 1993, lead was listed as a contaminant-of-concern at 24 Superfund sites in New Jersey.[3] Commercial use of metallic lead includes munitions, x-ray shields, and solder. At least half of lead consumed worldwide goes into producing lead-acid batteries used in automobiles and for other industrial applications.[4] Two particular uses of lead, since banned, have contributed to wide-spread multimedia contamination; those sources being leaded gasoline and leaded paint.
Stressor-specific impacts considered Key impacts selected	Neurological, hematological, cardiovascular and renal The most sensitive low-level effect of lead in children is cognitive deficit. In adults, increasing evidence suggests a causal relation between long-term, low-dose exposure to lead and hypertension[4]. At higher doses, effects in children include anemia and encephalopathy. High-dose exposure in adults causes renal tubular damage and encephalopathy.
Exposure Assessment	

Exposure routes and pathways considered	Incidental ingestion of lead-contaminated soil and indoor dust. Ingestion of lead-based paint. Ingestion of lead-contaminated drinking water from private wells and municipal water supplies (note- municipal supply wells are sampled regularly for compliance with the federal Action Level of 15 μg/l). Inhalation of airborne lead particulates and consumption of lead in diet (both exposure pathways expected to be minor contributors to total exposure).
Population(s)/ecosystem(s) exposed statewide	The ubiquitous nature of lead contamination in major environmental media (air, water, and soil/sediments) as well as its historic presence in consumer products such as paint, ceramics, plumbing supplies, canned goods, etc. has effectively spared no one in NJ at least some measure (as reflected in blood lead) of exposure.
Quantification of exposure levels statewide	Blood lead measurement (BPb) is a widely accepted biomarker of both lead exposure and effect. There are no BPb reporting requirements for the general population in NJ, but the State requires testing/reporting in pediatric populations. See Appendix A for number of new cases of Pb poisoned (>20 ug/dl) children for years 1993 - 2000. In 1999, two-year olds had the highest incidence (263 new cases in a statewide two-year old population of approximately 100,000) of lead poisoning in the pediatric population. Preliminary data for the year 2000 provides information on the prevalence of elevated blood leads (10-19 ug/dl) for children in New Jersey. From a total of 130,604 children tested, 4,101 had a blood lead between 10-14 ug/dl, while 1,522 had a blood lead between 15-19 ug/dl.[16]
Specific populations at increased risk	Children represent specific populations at increased risk for the following reasons: 1) Developing nervous system more sensitive to the effects of lead pre- and post-natal. 2) Children absorb lead more efficiently than adults do. Postulated mechanisms include increased calcium demand (Pb piggybacks on the calcium transport system) during periods of bone growth, and less developed gastrointestinal barrier. 3) Greater exposure from contaminated soil/dust due to mouthing activity. Within the childhood population, the sub-population with low socio-economic status are at even greater risk due to a higher probability of living in housing with peeling lead-based paint and increased risk of poor nutritional status (which increases lead absorption). Finally, children that exhibit pica (consumption of non-food items) behavior are at heightened risk.
Quantification of exposure levels to populations(s) at increased risk (i.e., susceptible sub-populations)	Children represent sensitive sub-populations by virtue of experiencing increased exposure and greater sensitivity to neurological impairment at relatively low blood lead levels. See Appendix A for number of new cases of Pb poisoned (>20 ug/dl) children for years 1993 - 2000. In 1999, two-year olds had the highest incidence (263 new cases in a statewide two-year old population of approximately 100,000) of lead poisoning in the pediatric population.

	Preliminary data for the year 2000 provides information on the prevalence of elevated blood leads (10-19 ug/dl) for children in New Jersey. From a total of 130,604 children tested, 4,101 had a blood lead between 10-14 ug/dl, while 1,522 had a blood lead between 15-19 ug/dl.[16]
Dose/Impact-Response Assessment Quantitative dose/impact-assessment employed for each population considered	Historically, blood lead concentration (BPb) has served as a biomarker of both long-term exposure and effect. The relationship between environmental lead exposure and BPb has been explored through epidemiologic studies that derive a "slope" between exposure and BPb and through biokinetic modeling. The intake slope factor for ingested lead in children is approximately .16 ug/dl per ug/day[5,6]. This value is likely to vary considerably based upon the bioavailability of the ingested lead. Alternatively, to obviate the influence of bioavailability, an uptake (absorbed dose) slope factor derived from biokinetic modeling[7] has been estimated to be .36 ug/dl per ug/day. The impact of low-level lead exposure on cognitive function in children has been well studied.[4] The Centers for Disease Control (CDC) has established guidelines for the interpretation of BPb in children: < 10 ug/dl - child not considered to be lead poisoned. 10 - 19 ug/dl - repeat BPb, nutritional and educational intervention, possible environmental investigation. 20 - 44 ug/dl - environmental evaluation/remediation and medical evaluation, possible chelation. 45 - 70 ug/dl - medical and environmental intervention including chelation therapy. > 70 ug/dl - medical emergency. Utilizing an intake slope factor of .16 ug/dl per ug/day, chronic ingestion of 60 ug/day of lead in a child would result in a BPb of approximately 10 ug/dl. Adults absorb lead less efficiently than children and thus have a lower intake slope factor. Adults are also less sensitive to the neurological effects of low-level lead exposure, but an increasing body of evidence suggests that BPb as low as 10 ug/dl may result in a small increase in systolic blood pressure in male adults[8].

Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences	Assuming national background levels of lead in water (4 μg/l) , air (.15 ug/m3) and diet (6.5 ug/day), children residing in homes with greater than 400 ppm of lead in soil may experience a greater than 5% probability of exceeding a BPb of 10 ug/dl[7]. An upper bound estimate of background concentration for urban residential areas is 100 ppm (NJDEP). The number of residential properties with soil lead exceeding 400 ppm is unknown.
	See Appendix A for number of new cases of Pb poisoned (>20 ug/dl) children for years 1993 - 2000. In 1999, two-year olds had the highest incidence (263 new cases in a statewide two-year old population of approximately 100,000) of lead poisoning in the pediatric population. Preliminary data for the year 2000 provides information on the prevalence of elevated blood leads (10-19 ug/dl) for children in New Jersey. From a total of 130,604 children tested, 4,101 had a blood lead between 10-14 ug/dl, while 1,522 had a blood lead between 15-19 ug/dl.[16]
Assessment of severity, persistence, irreversibility and frequency of effect(s) Size of population(s) affected	Lead poisoning can range from subtle neurological effects (approx. 10 ug/dl) to life-threatening encephalopathy (greater than 100 ug/dl). Lead has a comparatively long residency time in the body (T 2 = 30 days) and is stored in the skeleton; consequently, poisoning is usually associated with chronic exposure scenarios. Lead encephalopathy typically results in irreversible effects; however, there is conflicting literature relating to the reversibility of mild cognitive deficits[4]. Children age 6 months to 7 years are the most susceptible sub-population. There are approximately 100,000 children within each one-year age group in NJ Additional risk factors include low socioeconomic status, pica behavior and interior lead-based paint. See Appendix A for number of new cases of Pb poisoned (>20 ug/dl) children for years 1993 – 2000). In 1999, two-year olds had the highest incidence (263 new cases in a statewide two-year old population of approximately 100,000) of lead poisoning in the pediatric population. Preliminary data for the year 2000 provides information on the prevalence of elevated blood leads (10-19 ug/dl) for children in New Jersey. From a total of 130,604 children tested, 4,101 had a blood lead between 10-14 ug/dl, while 1,522 had a blood lead between 15-19 ug/dl.[16]

Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps.	Low. The dose/response relationship for lead has been well studied. Blood lead serves as an indicator of lead exposure and effect. State mandated screening of the pediatric population at ages one and two years provides a rich data base for determining the magnitude of lead exposure, although lack of compliance with screening protocol, especially by pediatricians serving low-risk populations is a concern.
Potential for future changes in the underlying risk from this stressor	(+) Childhood blood lead levels have been on a long steady decline since lead was removed from gasoline and house paint in the 1970's. Some pockets (e.g., deteriorated lead-based paint in old housing) of concern remain that are currently being addressed through a combination of regulations and public health education; so further, albeit modest, reductions in blood lead levels are likely.

Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	(L) Since lead is mostly a cumulative poison, a catastrophic release is neither likely nor of great consequence.
Extent to which risks are currently reduced through in-place regulations and controls	High. The combination of state-wide pediatric screening along with regulations governing the amount of lead in ambient air, drinking water, interior paint and gasoline have been very effective in reducing environmental lead exposure.
Relative contributions of sources to risk	
Allocation of stressor-specific risk to primary NJ sources	(L/M). The 1996 NJ air toxics inventory reports total lead emissions of 5.3 tons. For lead air monitoring up to and including the year 1996, NJ has exceeded the NAAQS for lead only once since 1992.[9]
Large business/industry	(L) Steel and Iron works were the largest category contributing 3 tons of the 5.3 tons total emissions reported by the air toxics inventory in 1996. Coal-burning utilities were the next largest contributor at .8 tons
Small business/industry	Auto body refinishing shops and industrial boilers are prevalent but low level contributors to lead emissions.[10]
Transportation	(M) The phasing out of leaded gasoline in the 1970's and 1980's drastically reduced the lead emissions associated with transportation. Small amounts of lead are still emitted from the burning of diesel fuel. Associated with transportation is the use of lead-acid batteries. Improper disposal and inappropriate recycling methods (e.g., unregulated "battery cracking" operations) can result in release of significant amounts of lead into the environment.

Residential	(L) Operation of residential coal/oil burners and open burning of trash releases small amounts of lead into the environment. The flaking of lead-based exterior paint is a source of soil lead contamination.

Agriculture	(L) Lead arsenate was used at the rate of several pounds per acre in apple and other fruit orchards, vegetable fields, golf courses and turf farms in NJ until 1967.[11] Leaded gasoline is still permitted for use in farm equipment[12] but leaded gasoline has not been produced in the U.S. since 1991.[4]
Recreation	(L) Firing ranges and hunting with leaded ammunition.
Resource extraction	(L) There are no major lead mining operations in New Jersey. There are 46 facilities in New Jersey that either manufacture or process lead.[2]
Government	(L) Lead munitions at military bases.
Natural resources	(L) The naturally occurring background lead concentration in New Jersey soil is approximately 5 - 30 ppm.[13]
Contaminated sites.	(L/M) A review of NPL sites in EPA Region II revealed that 22 sites in New Jersey had significant lead contamination.[14]
Diffuse and non-NJ sources	
Sediment	L/M - Sediments contain considerably higher concentrations of lead than corresponding surface waters[4].
Soil	M/H - The natural lead content of soil derived from crustal rock typically ranges from <10 - 30 ppm. However, soil adjacent to heavily traveled roadways may have 30 - 2,000 ppm lead, the result of past use of leaded gasoline[4], although the concentrations decrease exponentially up to 25 meters from the roadway[15]. Homes painted with exterior lead based paint may have lead concentrations greater than 6,000 ppm with the highest concentrations typically around the house foundation[4]. Additionally, there are many lead-painted components of the infrastructure, including bridges, water towers and trestles that release lead secondary to weathering.

Non-local air sources (including deposition)	L - This is a minor source - the NAAQS for lead (1.5 ug/m^3) has not been exceeded in New Jersey since 1992[9].

Biota sinks	L - Lead does not bioconcentrate in fruits or vegetables. Leafy green and root vegetables may have elevated lead concentrations but much of the lead is associated with surficial soil contamination. Animals, like humans, store lead in their skeleton however, scarce data limit the ability to estimate the impact on the environment.

Human Health Issue Summary: Lead

What is it?
Lead is a naturally occurring metal used in a wide range of industrial and commercial applications. Two particular uses of lead, which have since been banned, have contributed to widespread environmental contamination: leaded gasoline, and leaded paint. Small amounts of lead continue to be emitted in diesel exhaust, and the majority of ongoing industrial emissions are attributed to steel and iron works. Coal burning power plants also emit quantities of lead. Human health effects arise as a result of exposure to historic concentrations of lead in the paint used in older homes, and in the soils adjacent to roadways and lead-painted structures. These can range from subtle neurological effects, such as a learning deficit, to anemia and life-threatening encephalopathy at higher exposures. There may also be a link between long term exposure and hypertension in adults. Lead accumulates in soils, surface waters, and sediments presenting a toxic hazard to fish, amphibians, reptiles, birds, and mammals.

What's at risk?
Because of the pervasiveness of lead in the environment, exposure of people and wildlife occurs statewide. Children are far more likely than adults to ingest contaminated soil or peeling paint, moreover, their bodies absorb it more efficiently and their developing nervous systems are more sensitive to its effects. Although contamination is often greatest in urban/suburban regions, elevated lead levels are found in soils, sediments, and surface waters throughout the state.

What are the human health impacts in New Jersey?
There are no requirements for testing the general population for lead exposure, but New Jersey requires testing of children under 7. The Centers for Disease Control considers child blood lead levels > 10 micrograms per deciliter of blood to be elevated, and children with levels >20 ug/dl are considered lead poisoned. In 1999, there were a total of 802 cases of lead poisoning in children under 7 in New Jersey. Preliminary data for 2000 indicates 4% of children tested had elevated blood lead levels. Since 1993, New Jersey has documented more than 15,000 cases of lead poisoning in children.

What's being done?
The phasing out of leaded gasoline has drastically reduced lead emissions to the air. Regulations restrict the amount of lead in the air, in drinking water, and in consumer products. A number of laws also govern the cleanup of contaminated sites. Public health education, along with statewide pediatric screening has also contributed to reductions in blood lead levels.

Issue: Lead
Author: Mark Maddaloni
Version: 01/02/01

Severity of specified health effects at current levels of exposure	Size of population at significant risk for each health effect	Are there more discrete communities at elevated risk?	Overall risk ranking
H (4)	M **(3)**	**Y (5)**	**H (4)**

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Issue: Lead
Author: Mark Maddaloni
Version: 01/02/01

Appendix A

First Report for Children with Blood Lead Test Results Greater Than or Equal to 20 ug/dl
Reported to NJDHSS January 1, 1993 through October 31, 2000

TABLE OF AGE BY YEAR

Age	1993	1994	1995	1996	1997	1998	1999	*2000	Total
0	46	24	26	15	18	13	16	1	159
1	656	605	390	368	318	275	198	134	2944
2	1115	974	565	482	463	365	263	221	4448
3	1006	705	433	337	308	212	150	138	3289
4	748	547	331	227	190	168	107	79	2397
5	406	303	181	116	94	85	40	46	1271
6	205	130	61	49	42	28	28	20	563
Total	4182	3288	1987	1594	1433	1146	802	639	15071

- year to date (10/31/00

Issue: Legionella
Author: Tom Atherholt
Version: 02/21/01

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Legionella** *Legionella pneumophila* (mostly serogroup 1) and other *Legionella* bacteria. Twenty two species are known to be pathogenic to humans (Fujii and Yoshida, 1998). In addition, a non-culturable group called *Legionella*-like amebal pathogens (LLAP) may also cause respiratory disease in humans (Adeleke et al., 1996).
Description of stressor (including etiology)	*Legionella pneumophila* and related bacteria are ubiquitous, free-living (not pollution-associated) bacteria. Some species are potential human pathogens. They typically reside within several types of free-living protozoan microorganisms (Fujii and Yoshida, 1998). In fact, *Legionella* are unique among bacteria in that not only can they survive within protozoa (as well as within protozoan cysts), but many species can also multiply within the protozoa (Kwaik *et al.*, 1998). Within humans, they reside within alveolar macrophages (Fields, 1997). Under certain, usually man-made environmental conditions, these bacteria and their hosts can multiply to high numbers. These conditions include warm waters (25 - 42 ^{o}C; CDCa or 32 - 45 ^{o}C; CDCb), stagnant waters having excessive scale and sediment, and waters containing the presence of certain free-living amoeba capable of supporting the growth of *Legionella* (CDCa). Inhalation of aerosolized droplets containing high numbers of these bacteria can cause a flu-like disease called Pontiac fever (PF) or a more serious, sometimes fatal type of pneumonia called Legionnaires' disease (LD, or legionellosis), in susceptible individuals. Most nosocomial (hospital-acquired) cases of LD occur year-round while most (but not all) LD outbreaks occur during the summer or early fall (CDCb, Hall, 1999). The *Legionella* bacterium was first recognized as a human pathogen in 1977 following an outbreak of pneumonia among American Legion convention delegates in Philadelphia in 1976 (Roy, 1999). Diagnosis of legionellosis is often difficult (Stout and Yu, 1997) and there is a comparatively high mortality rate associated with certain risk groups (see below).
Stressor-specific impacts considered including key impacts	Inhaled *Legionella pnuemophila* and/or related bacteria can cause, in susceptible individuals, one of two types of legionellosis: Legionnaires' disease (a serious, sometimes fatal pneumonia) or a milder disease called Pontiac fever (a flu-like illness). Legionnaires' disease symptoms include high fever, chills, aches and pains, dry cough, chest pain, and difficulty breathing. Some patients experience muscle aches, headache, tiredness, loss of appetite, or diarrhea

	(CDCb). A few cases involve delirium and some cases are fatal due to respiratory or multi-organ failure (Stout and Yu, 1997). PF symptoms include fever and muscle ache without pneumonia.
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	Inhalation of water aerosols containing high concentrations of *L. pneumophila* bacteria. Waterborne ingestion of low levels of *Legionella* is common, perhaps universal, but is not associated with disease. Inhalation of aerosolized droplets of water containing potentially harmful levels can occur by being in close proximity to certain potential sources such as showers, faucets, cooling towers, evaporative condensers, whirlpool spas, respiratory therapy equipment and room air humidifiers in which *Legionella* have multiplied to high levels following the multiplication of their protozoan hosts (CDCb; Brieman and Butler, 1998; Lin *et a.*, 1998; Rohr *et al.*, 1998). Recently, several cases of legionellosis due to the species *Legionella longbeachae* have been documented in persons exposed to potting soil during gardening activities. The urinary antigen test normally used to detect exposure to legionellae will not detect this species (MMWR, 2000). Exposure does not occur by person-to-person transmission and auto or household window air-conditioners have not been implicated in reported cases (CDCb).
Population(s)/ecosystem(s) exposed statewide	Anyone has the potential to be exposed to unsafe levels. Most exposed healthy individuals do not become sick. For persons with normal immune system function, a high dose of *Legionella* is believed to be required for illness to occur (Fujii and Yoshida, 1998). Of exposed individuals who become ill, most have weakened immune systems or underlying illnesses (see below). Hence *Legionella* are considered opportunistic pathogens.
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	Quantification is difficult and unreliable (Boulanger and Edelstein, 1995). In addition, *Legionella* often enter a viable but non-culturable state in environmental waters (Steinert *et al.*, 1997). Furthermore, they usually reside within free-living protozoa (Fujii and Yoshida, 1998). It has been proposed that the infectious particle for Legionnaires' disease is an amoeba infected with *Legionella* bacteria (Kwaik *et al.*, 1998). Therefore, published counts of *Legionella* bacteria in environmental waters, based on *in vitro* culture or direct microscopic examination,

	may be of limited health relevance. The Centers for Disease Control and Prevention (CDC) does not currently recommend routine culturing of water systems for *Legionella* (CDCa), because there is no dose - health risk information to interpret such data (see footnote 1). *Legionella* are commonly found in natural waters including ground waters (Lye *et al.*, 1997), as well as a variety of man-made aquatic habitats as described above (Fliermans, 1996; Lin *et al.*, 1998). They are present at low, often undetectable levels (by culture) in treated potable waters that supply homes, offices, hospitals, etc. (Muraca *et al.*, 1988), but can multiply under warm water and other favorable growth conditions.
Quantification of exposure levels statewide, including populations at significantly increased exposure (con't)	Using direct fluorescent antibody (DFA) enumeration techniques, Tison and Seidler, (1983) reported the following concentration ranges: untreated drinking water sources, 1.4×10^3 - 1.7×10^4 cells/100 ml; chlorinated water, $< 8 \times 10^2$ - 1.4×10^3 cells/100 ml; slow sand filtered and chlorinated water, $< 5.4 \times 10^2$ - 4.6×10^3 cells/100 ml; water treated by flocculation, filtration and chlorination (conventional treatment), $< 8 \times 10^2$ - 2.2×10^3 cells/100 ml. Other investigators found lower concentrations (Ellis, 1993). Zacheus and Martikainen (1996) observed a culturable concentration range of *Legionella* in hot water systems, where these bacteria (as well as their protozoan hosts; Rohr *et al.*, 1998) have been shown to multiply, of 3×10^2 - 3.5×10^4 colony forming units (CFU) per 100 ml. A recent survey of hospital cooling tower water samples found a *Legionella* concentration range of 10^1 - 10^5 CFU/100 ml (Miyamoto *et al.*, 1997). A number of early legionellosis disease outbreaks involved contaminated cooling tower (CT) waters (Fujii and Yoshida, 1998). Counts in these waters were in the range of 10^2 to 10^8 CFU/100 ml (Ellis, 1993)(see footnote 2). Counts in tap waters in hospitals with Legionnaires' disease cases were in the range of $1\text{-}5 \times 10^5$ CFU/100 ml (Ellis, 1993). Counts > 10^5 CFU/100 ml were found in a whirlpool spa water following a legionellosis outbreak on a cruise ship (Jernigan *et al.*, 1996).
Specific population(s) at increased risk	Persons with weakened immune systems are at greater risk since most exposed healthy individuals do not become sick. Mortality rates in otherwise healthy persons is very low (Edelstein, 1995). The elderly appear to be at increased risk (Jernigan *et al.*, 1996) with most cases occurring in the 40-70 age group and with a male:female case ratio of 2 or 3:1(Ellis, 1993). Other risk groups include heavy smokers and those with underlying diseases of the heart, lungs, kidneys or chronic diabetes. Dialysis patients and surgical, especially transplant patients, are also at increased risk (Stout and Yu, 1997). Also at increased risk are persons whose immune systems are suppressed by diseases such as cancer and AIDS. Those that take drugs that suppress the immune system (*e.g.*, organ transplant patients or persons taking systemic steroids) are also at higher risk (CDCb). Nosocomial LD has also been reported among patients at children's hospitals (CDCa). Fatality rates are highest for immunosuppressed patients or those with underlying

	diseases (Edelstein, 1995).
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	Same as for statewide population.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	None. Berendt *et al.,* (1980) conducted a *Legionella* aerosol challenge experiment using male Hartley strain guinea pigs. The LD_{50} (dose lethal to 50% of the test animals) aerosol dose was 1.4×10^5 viable cells while the ID_{50} (the infectious dose) dose was < 129 viable cells, the lowest dose administered. However, it is not known if the guinea pig model is valid as a predictor of human exposure dose response. Beyond this, the data are insufficient for dose-response modeling (Rusin *et al.*, 1997).
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	An infectious dose for humans has not been defined.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Pontiac fever = low severity, reversible symptoms, low frequency of occurrence. No mortality. Legionnaires' disease = low to high severity, some mortality but otherwise reversible symptoms, low frequency of occurrence.
Size of population(s) affected	During outbreaks, the attack rate for Legionnaires' disease is low (0.1-4 %) while that for Pontiac fever is high (90%) (Fliermans, 1996). The CDC estimates that 8,000 to 18,000 people get Legionnaires' disease in the United States each year (CDCb). Much of this exposure probably occurs indoors, but there is some outdoor exposure as well. Ten to 20% of cases can be linked to outbreaks, the rest are sporadic cases (CDCb). Mortality is estimated to be 5 to 15 or 30% of Legionnaires' disease cases (CDC provides conflicting estimates; CDCb). Case rates in the U.S. between 1988-1998 ranged from 0.4 - 0.6 per 100,000 population (CDC, 1999). Assuming affected persons are randomly distributed (there is no information to support this assumption), then in New Jersey the yearly number of affected persons is estimated to be 237 - 533 (CDCb) (see footnote 3). The number of NJ deaths associated with disease is estimated to be 12 - 15 per year. Reported cases of legionellosis in NJ, from 1993-1996, averaged 33 cases per year (0.4 per 100,000 population). This number is in agreement with information stating that that only 5%- 10% of estimated cases are reported

	(CDCb). Illness and fatality rates are likely to vary from year to year. 1996 NJDHSS data show that no cases were under the age of 30 (Fishman *et al.*, 1999) and U.S. data show very few cases in people < 30 years old (CDC, 1999). The occurrence of Pontiac fever is estimated to be 2 to 100 times more frequent than Legionnaires' disease (Hall, 1999).
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	H - Size of NJ population adversely affected is an estimate only.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	Low.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	0
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	Low. Erythromycin or azithromycin, often given in conjunction with other antibiotics, is effective in treating *Legionella* infection (Klein and Cunha, 1998; Stout and Yu, 1997; Edelstein, 1995; Nechwatal *et al.*, 1993). Thus, the prospect of a large-scale outbreak with serious sequelae is remote. Nevertheless, *Legionella* infection is difficult to detect and, even when detected and treated with antibiotics, some mortality still occurs (Fliermans, 1996; Nechweatal, 1993).
Extent to which risks are currently reduced through in-place regulations and controls	*Legionella* are specified as one of the pathogenic microbes controlled, through disinfection and filtration, by the USEPA Surface Water Treatment Rule (USEPA, 1989). The SWTR is adopted in NJ by reference, hence all public surface water treatment facilities in the State comply with the SWTR requirements. In addition, for ground water source systems, NJ regulations require that community water systems (but not non-community water systems) disinfect their water. However, ingestion of drinking water is not considered to be significant direct source of infection, *Legionella* and their protozoan hosts are fairly resistant to disinfection (Fliermans, 1996; Ellis, 1993) and concentrations of *Legionella* may increase to potentially harmful levels in hot water systems or in other man-made aquatic environments (see above). The NJ Department of Health and Senior Services (DHSS) has several regulations aimed at the control of *Legionella* (NJAC 8:57; 13.4(c)). Legionellosis is a reportable disease in NJ (Fishman, 1999) meaning that physicians, hospitals and other health care providers are required to report all known cases of the disease to the DHSS. Indoor air quality regulations control building air handling equipment maintenance as well as regulations specifically addressing microbial contamination (NJAC 12:100-13).

	Legionella growth in water systems can be controlled through the implementation of preventative procedures (CDCb; see footnote 4).
Extent to which risks are currently reduced through in-place regulations and controls (con't)	Copper-silver ionization units have also been shown to control *Legionella* in some (Stout and Yu, 1997; States *et al*., 1998) but not all (Kioski *et al.*, 1997) hospital or nursing home water distribution systems. Hot-water tank temperatures should be maintained between 71 and 77 ^{o}C (Hall, 1999). At 66^{o}C, *Legionella* are killed within 2 minutes (Fujii and Yoshida, 1998). The Food and Drug Administration has cleaning and maintenance guidelines for the use of ultrasonic vegetable misters (Hall, 1999).
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Hospitals	Medium. 23% of all cases of Legionnaires' disease are nosocomial (hospital-acquired). Highest percentage of mortality occurs during nosocomial outbreaks (CDCb).
Large business/industry	Low. Air conditioning cooling towers, contaminated hot water systems.
Small business industry	Low. Air conditioning cooling towers, contaminated hot water systems.
Transportation	None.
Residential	Medium. Contaminated hot water systems (via showers, faucets), whirlpool spas, room-air humidifiers, respiratory therapy equipment.
Agriculture	None.
Recreation	Low. Contaminated hot water systems in hotels or public buildings, whirlpool spas in hotels and cruise ships.
Resource extraction	None.
Government	Low. Contaminated hot water systems, cooling towers.
Natural sources	High. All *Legionella* are ultimately derived from natural sources via fresh water or potable water.

Issue: Legionella
Author: Tom Atherholt
Version: 02/21/01

Contaminated sites	None.
Diffuse and non-NJ sources	None.
Sediment	Medium ? Possible source for waterborne organisms?
Soil	Low LD outbreaks near construction sites initially believed to be due to dispersed soil, but the organism does not survive dry environments. Construction outbreaks now believed to be due to massive descaling of plumbing systems during construction (Fields, 1997).
Non-local air sources (including deposition)	None.
Biota sinks	High. *Legionella* are obligate parasites of free-living protozoa.

Human Health Issue Summary: Legionella

What is it?
Legionella is a group of bacteria, some of which are known to be pathogenic to humans. Under natural conditions, Legionella bacteria do not pose a threat. In certain (primarily indoor) conditions, they can multiply to unsafe levels. Humans may become exposed via inhalation of contaminated aerosols that arise from stagnant warm water found in indoor air handling systems. Inhalation of high numbers of these bacteria can cause a flu-like disease called Pontiac fever, or a more serious and sometimes fatal type of pneumonia called Legionnaire's disease, first recognized in 1977 following an outbreak of pneumonia at an American legion convention in Philadelphia.

What's at risk?
Anyone has the potential to become exposed, but most healthy individuals will not become ill. People with an existing illness are more likely to become ill as a result of exposure. Smokers, the elderly, chemotherapy patients, and individuals with weakened immune systems are examples of more susceptible groups. Most cases have occurred in the 40-70 age group.

What are the human health impacts in New Jersey?
Reported cases of legionellosis in New Jersey from 1993-1996, averaged 33 cases per year. However, it is likely that only 5-10% of cases are reported. Based on Centers for Disease Control statistics, an estimated 237-533 people may contract legionellosis each year in New Jersey, with potentially 12-15 deaths resulting. Fatality rates are highest for immune-suppressed patients, or those with underlying disease. The occurrence of Pontiac fever is estimated to be 2 to 10 times more frequent than legionellosis.

What's being done?
Growth of the bacteria can be controlled through the implementation of preventative procedures. Indoor air quality regulations apply to air handling equipment, and address microbial contamination specifically.

Issue: Legionella
Author: Tom Atherholt
Version: 02/21/01

Severity of specified health effects at current levels of exposure (H,M,L)	Size of population at significant risk for each health effect (H,M,L)	Are there discrete communities at elevated risk? (Y,N)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L)
L, 2	L, 1	Y	L, 2
			L, 2

My overall ranking is based on the fact that there is a small estimated annual mortality in NJ of 12-15 per year, predominantly in predisposed individuals. It should be noted that illness and fatality rates are likely to vary from year to year and that the "NJ disease burden" assumes a random distribution of national case estimates which is not likely to be the case.

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Footnotes:

(1) One school of thought, regarding hospitals in which there are no identified Legionnaires' disease cases, suggests that routine monitoring of the potable water system be conducted and, when > 30 % of the tap water samples test positive for Legionella, the water system should be decontaminated and active surveillance for legionellosis cases initiated (CDCa). However, in the absence of legionellosis, the relationship between the results of the water cultures and the risk of legionellosis remains undefined. Legionella bacteria are frequently found in hospital water systems (Lin *et al.*, 1998) without any evidence of legionellosis and the risk of disease depends on other factors besides the presence or amount of Legionella in a water supply (CDCa). These factors include the degree to which contaminated water is aerosolized into respirable droplets, the proximity of the aerosol to potential host, the susceptibility of the host, and the virulence properties of the contaminating strain (CDCa).

(2) The Ministry of Health and Welfare, Japan, has issued guidelines for the remediation of contaminated cooling tower waters based on viable counts of the Legionella bacterium in such waters. According to the guideline, viable counts are classified into four ranges: permissive ($<10^2$ CFU/100 ml), observational range (between 10^2 and 10^3 CFU/100 ml), careful observational range (between 10^3 and 10^5 CFU/100 ml) and emergency level (10^5 and higher CFU/100 ml). The emergency level requires instant physical and chemical cleaning of the cooling tower system (Fujii and Yoshida, 1998). However, the relationship between these levels in cooling tower water and the risk of Legionnaires' disease is not known.

(3) US population at year 2000 ~ 270 million. NJ population at year 2000 ~ 8 million (8 / 270 million = 0.03).

(4) (a) potable water at outlets should be either greater than or equal to () 50 ^oC or less than 20 ^oC; (b) a 1 -2 mg/L concentration of free chlorine in the tap water may also help control the growth of Legionella; (c) nebulizing equipment should be filled using only sterile water and the use of large-volume room-air humidifiers is discouraged unless they are sterilized or subjected to high-level disinfection daily and filled only with sterile water; (d) contaminated water systems can be decontaminated by superheating the water in the distribution system (5 minute flushing of each faucet with 65 ^oC water) or by hyperchlorination (5 minute flushing of each tap with water containing 10 mg/L free chlorine). Scalding warning signs should be posted if hot water decontamination is employed. Water heaters and hot-water storage tanks should be periodically cleaned to remove scale and sediment.

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Lyme Disease** In the Northeast, Lyme disease (LD) is caused by the spirochete *Borrelia burgdorferi* transmitted through the bite of infected black-legged ticks, *Ixodes scapularis* (Acari: Ixodidae). This tick is also responsible for transmitting the agents that cause human granulocytic ehrlichiosis (*Ehrlichia phagocytophila*) and human babesiosis (*Babesia microti*). The tick *Amblyomma americanum* is responsible for transmitting the spirochete *B. lonestari*, the agent that is purported to cause a Lyme disease-like illness. Other tick-borne diseases in NJ include Rocky mountain spotted fever (*Rickettsia rickettsii* transmitted by *Dermacentor variabilis*) and human monocytic ehrlichiosis (*E. chaffeenensis* transmitted by *A. americanum*). Because of its significant public health impact, this review will be limited to LD.
Description of stressor (including etiology)	Lyme disease essentially has a global distribution, including North America, Europe, the former Soviet Union, China, and Japan. In these regions, the *Borrelia* causative agents, which vary geographically, are transmitted by one of several tick species belonging to the *I. ricinus* complex. In the US, LD has been identified in 47 states. Endemic foci occur in the Northeast, the upper Midwest, and the West, with the majority of cases occurring between Massachusetts and Maryland. In the Northeast and upper Midwest, the vector is *I. scapularis*, while in the West the vector is the western black-legged tick, *I. pacificus*. LD was first described in the US in Connecticut in 1975, while the first case in NJ was reported in 1978. Since then, LD has emerged as a significant public health threat.
stressor-specific impacts considered including key impacts	LD is a multi-systemic, inflammatory disease often characterized in its early phase by a distinctive skin lesion known as *erythema migrans* (EM), which first appears as a red, raised area, but tends to expand in size over time, and may develop centralized clearing. Single or multiple lesions are often accompanied or preceded by a variety of other signs and symptoms including headache, fever, malaise, joint pain, stiff neck, and nausea. The incubation period is 3-30 days. Within weeks or months of the appearance of EM, neurologic or cardiac symptoms may appear. Some individuals may experience arthritic manifestations marked by swelling and pain in the large joints, particularly the knees, within weeks or years of onset. Symptoms are often recurrent and may become chronic in untreated individuals. NJ reports ca. 2000 cases annually and consistently ranks among the top 5 states with respect to the number of confirmed cases reported each year. Pets, particularly canines, and livestock may also be impacted by LD. The impact of LD on wildlife is unknown.
Exposure Assessment	
Exposure routes and pathways considered **(include indoor air as appropriate)**	LD results primarily from the bite of an infected tick. Ticks acquire *B. burgdorferi* after feeding on infected hosts as subadults. The infection is transmitted to other hosts during subsequent feedings. The white-foot mouse,

	Peromyscus leucopus, is considered the primary reservoir. There is no risk of human-to-human transmission.
Population(s)/ecosystem(s) exposed statewide	Everyone in NJ is potentially exposed to the bite of an infectious tick. LD cases have been reported from each of NJ's 21 counties. The risk is greatest among populations residing in wooded suburban and rural environments. Individuals with occupational or recreational exposure to wooded areas are also at risk. Similarly, pets and livestock contacting wooded environments are at risk. A LD vaccine is currently available for individuals aged 15-70 years.
Quantification of exposure levels statewide, including populations at significantly increased exposure **(include indoor air as separate category as appropriate)**	LD is considered endemic throughout NJ. Historically, most LD cases during the 1980s were reported from Monmouth and Ocean Counties. During the 1990s, the majority of cases were reported from Hunterdon, Morris, Somerset, and Warren Counties. However, wooded suburban and rural environments are better predictors of risk than political boundaries. LD seems to be most prevalent in wooded areas (tick habitat) experiencing significant residential development. In such situations, individuals may be exposed on a more-or-less continuous basis. Minimum Infection Rates (MIR) for *I. scapularis* adults and nymphs range between 40-50% and 20-25%, respectively. Since there is virtually no transovarial transmission, larval ticks are rarely infected.
specific population(s) at increased risk	Individuals in frequent contact with tick habitat are at increased risk. A large proportion of cases occur among children, presumably because of increased exposure and infrequent use of preventive measures.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) **(include indoor air as separate category as appropriate)**	Specific quantification is not possible at this time. Key components to consider for quantification include tick population density, time spent in high-risk habitat (as tick populations throughout NJ are infected with *B. burgdorfer*) and duration of tick attachment. Little transmission occurs within the first 24 hours of attachment. A model estimating the probability of infection from individual ticks removed from patients within a LD-endemic area yielded an overall probability of 4.6%.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	N/A
Risk Characterization	
risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	On a broad spatial scale, relative LD risk is monitored through human case surveillance. Using surveillance data and census information, case rates (no. cases/ 100,000 population) can be calculated for the state and specific counties and/or municipalities. However, caution should be exercised when assigning transmission risk. Human surveillance data may be skewed by reporting artifact and even in areas hyperendemic for LD, risk will vary significantly based on frequency of exposure to tick habitat and preventive measures employed. That being stated, Hunterdon, Morris, Somerset, and Warren Counties currently report case rates significantly higher than the overall NJ case rate.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Diagnosed and treated early, the effects of LD are generally mild and transient. Misdiagnosed or undiagnosed/untreated cases may result in more severe complications, including cardiac, neurologic, or arthritic manifestations. Left untreated, these manifestations may become chronic and marked by recurrent episodes. *Borrelia*-related damage to joints, for example, may be irreversible. The proportion of asymptomatic cases is unknown. LD is not generally considered fatal.

size of population(s) affected	An average of 2,000 confirmed LD cases are reported each year in NJ's population of 8.4 million.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	A confirmed LD case is one that meets a restrictive CDC case definition. Confirmation of cases is used primarily to standardize data for surveillance purposes so that trends in incidence can be more accurately tracked. The number of confirmed cases is thought to represent only a portion, perhaps as low as 10%, of the number of LD cases that are actually diagnosed and treated each year. This disparity results from a combination of underreporting by physicians, absence of (or failure to recognize) signs/symptoms needed to meet case definition, prompt treatment, and lack of a confirmatory test.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	Since LD has been a reportable disease in NJ for 2 decades, observed trends can be expected to continue. Absent major shifts in residential development or availability of confirmatory tests, the incidence of LD in NJ can be expected to remain at similar levels.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	Educate public to prevent exposure: ++ Improve human surveillance: + Implement tick assessment/management programs: ++ Develop a confirmatory test for LD: ++ Identify environmental factors impacting the LD transmission cycle: ++
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	On a large spatial scale, a series of severe winters could temporarily impact risk resulting from tick and host mortality. Similar trends, albeit on smaller spatial scales, could be observed following major forest fires that occur with some frequency in the Pine Barrens, for example. Major shifts in host density, such as increases or decreases in deer harvest, or disease outbreaks affecting the deer herd, might also have a temporary effect.
extent to which risks are currently reduced through in-place regulations and controls	Effective public education and surveillance are the extent of current intervention. No organized tick assessment or management programs have been established. Any tick control is performed either by homeowners or PCOs.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
large business/industry	L
small business industry	L
Transportation	L
Residential	M create/enhance host habitat (woodpiles, landscaping)

	or survival (bird feeders, feed deer); in wooded areas, frequent exposure to ticks
Agriculture	L
Recreation	M in wooded areas, frequent exposure to ticks
Resource extraction	L
Government	L
natural sources	M tick-host interaction/transmission cycle most significant in such areas
Contaminated sites	L
diffuse and non-NJ sources	
Sediment	
Soil	
non-local air sources (including deposition)	
biota sinks	

Issue: Lyme Disease
Author: Terry Schulze
Version: 04/19/01

Human Health Issue Summary: Lyme Disease

What is it?

Lyme disease is a multi-systemic, inflammatory disease transmitted through the bite of infected ticks. Diagnosed and treated early, the effects are generally mild and transient. Misdiagnosed or untreated cases may result in more severe complications, including cardiac, neurologic, or arthritic conditions. Individuals may experience the onset of these symptoms anywhere from weeks to years from the time of infection. Damage to joints can be irreversible if left untreated. White-tailed deer (also evaluated as a biological stressor) are known to carry the tick that spreads the disease.

Who's at risk?

Specific populations at risk are those living or working in wooded suburban or rural environments in New Jersey. However, Lyme disease cases have been reported in all New Jersey counties. A large proportion of cases occur among children, presumably because of increased exposure and infrequent use of preventive measures.

What are the human health impacts in New Jersey?

An average of 2,000 cases are reported annually (approximately 24 cases per 100,000 population). New Jersey consistently ranks among the top 5 states with respect to the number of confirmed cases reported each year.

What's being done?

A vaccine is currently available for individuals aged 15-70 and public education and surveillance help to reduce the risk of exposure. No organized tick assessment or management programs have been established. Efforts to control the deer population, if successful, will also reduce the potential for infection.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	**Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)**	**Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)**	**Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)**
L,1 (in properly diagnosed and treated individuals) L,5 (in mis-/undiagnosed/ treated individuals)	L, 2	Y, 4 (Individuals with frequent residential, occupational, and recreational exposure to tick habitat)	L, 2
			L, 2

Issue: Mercury
Author: Alan Stern
Version: 12/18/00

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Mercury** Mercury (Hg) - including Hg^0, ionic/inorganic (largely Hg^{++}), and organic (largely methylmercury (MeHg)
Description of stressor (including etiology)	Mercury is a metallic element. The metallic form, Hg^0, is a silvery liquid which has a significant volatility at room temperature. In the vapor form it can be inhaled. It is used in thermometers, various electrical switches, and pressure gauges, and in connection with Latin American and Caribbean folk/religious practices (e.g., Santeria) where it may be burned with a candle, atomized with perfume, etc.[1] It is also used in most dental amalgams (fillings), and can release vapor into the mouth which is then inhaled. Hg^{++} can be found associated with various anions. Of the most common salts, $HgCl_2$ is water-soluble and has been found as a contaminant in NJ private well water. Although the source of the contamination is not definitely known, it appears to be due, at least in part from use as a pesticide in agriculture and landscaping. [2.] HgS is highly insoluble and functions as a long-term sink for environmental Hg. MeHg is formed by bacterial metabolism of Hg^{++} in aquatic systems. It is highly bioaccumulative in aquatic biota and has been found at levels >2 ppm in 14% of tested water bodies in NJ. [3] Humans are exposed by ingestion of contaminated fresh water and marine fish (including commercial fish). It largely originates from atmospheric deposition of Hg^{++} resulting from fossil fuel and waste combustion, as well as numerous small sources[3], and historically from direct discharge to specific waterbodies.
Stressor-specific impacts considered	Hg^0 - Neurotoxicity [5] Hg^{++} - Kidney toxicity[5] MeHg - Developmental neurotoxicity (i.e., effects on the developing fetus) resulting in subtle deficits in cognitive and motor functions.[5]
Key impacts selected (critical health/ecological effects)	(Note, these are effects which are seen at relatively low levels of exposure. More severe, less subtle and more debilitating effects occur at higher levels of exposure). Hg^0 - tremors, coordination subtle deficits, deficits in neurobehavioral performance. Possible subtle

	neurodevelopmental effects on behavior of children exposed during pregnancy to Hg^{++}. Hg^{++} - glomerular autoimmune effects, glomerular and tubular inflammation, tubular atrophy, changes in kidney weight. [5,6] MeHg - Subtle developmental deficits in neurobehavioral, and neurophysiological function (e.g., cognition, attention, motor function) in children exposed *in utero* [6]
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	Hg^0 -inhalation of indoor air in residences from inhalation of vapor inside residences resulting from spills and intentional releases (associated with folk/religious practices); inhalation of Hg^0 released from dental amalgams. Hg^{++} - consumption of contaminated private well water[1] (note - municipal supply wells are sampled regularly, and detection of Hg^{++} at concentrations above the Maximum Contaminant Level (2 μg/l) results in elimination of that source). MeHg - maternal consumption of contaminated NJ freshwater and marine fish, and commercial fish from various sources (including imports) during pregnancy. [7]
Population(s)/ecosystem(s) exposed statewide	Hg^0 - There are currently no data from NJ on the number of people exposed to Hg^0 in dwellings, or the number of dwellings contaminated with Hg^0. Only preliminary data on the extent of use of Hg^0 in folk/religious practices exists from non-random sampling of dwellings in other parts of the U.S. In N.Y. City, survey of 203 Latin American and Caribbean adults in selected neighborhoods, 44% of Caribbean and 27% of Latin American respondents stated that Hg^0 is used in their homes, cars or carried on their persons [1]. In Hartford, CT, some evidence of mercury use was found in 14% of surveyed homes of Hispanics [1]. Because of the non-random nature of these samples, because their relevance to NJ is unclear, and because the nature and extent of the cultural and ethnic association is not known, extrapolation of these values to estimates of persons exposed in NJ is not appropriate. In addition, several buildings in which Hg^0 was used in manufacturing have been converted to residential use with subsequent finding of elevated Hg vapor in living spaces. Known cases have been remediated, and it is not known whether other such cases currently exist. In a 1998 study[12] of an adult U.S. military population (average age = 53 years), the average number of amalgam surfaces per individual was 19.9. Anecdotal information suggests, however, that there is a much lower frequency of dental amalgams for younger age groups as a result of fluoridation and fluoride treatments.
	Hg^{++} - Of 2,239 private wells tested in the Kirkwood-Cohansey aquifer system in NJ (mostly Ocean and Atlantic counties), 1,321 (59%) had detectable levels of Hg (0.2-0.5 μg/l, ~95% of total Hg as Hg^{++}), and 306 wells, 14% of the total tested had Hg levels exceeding the MCL (2 μg/l) [8]. Assuming approximately 3 persons per household, the total number of persons exposed in NJ to well water exceeding the MCL is likely to be greater than 1,000, but there is no clear basis for estimating a more exact number. Testing was not random, but was biased toward

	detection of Hg contamination, and the percent exceeding the MCL is, therefore, likely to overestimate the percentage of total wells in this aquifer system exceeding the MCL. Little data are available from other areas of NJ.
Population(s)/ecosystem(s) exposed statewide - continued	MeHg - Data from a telephone survey of fish consumption among NJ households indicates that 93% of NJ adults consume fish at least a few times per year [9]. In a study of Hg exposure of pregnant women in NJ, 92% reported at least some fish consumption [10]. There were 114,335 births in NJ in 1996 [11]. Therefore, over 105,000 infants born in NJ each year are exposed to some MeHg in utero. For the general population of adults in NJ, it is estimated that approximately 5% is exposed over the RfD intended to be protective against neurologic effects of MeHg in adults (0.3 μg/kg/day) [9]. For women of childbearing age/pregnant women in NJ, 10-21% are estimated to be exposed above the RfD intended to be protective against neurologic developmental effects to the fetus *in utero* [10]. This corresponds to approximately 11,000-24,000 infants per year in NJ.
Quantification of exposure levels statewide (include indoor air as separate category as appropriate)	Hg^0 - Unknown. However, in theory only a few drops of Hg^0 would be required to reach an unacceptable concentration of Hg in indoor air. Large spills and intentional releases could, therefore, produce indoor air levels which could result in adverse health effects with long-term exposure. WHO (1991) estimates the daily exposure to Hg^0 from dental amalgams at 4-21 : g/day. Hg^{++} - No community water systems in NJ had exceedances of the MCL in 1998. See below for consumers private well water. MeHg - all NJ adults –[9] percentile of population — av. daily dose (μg/kg/day) mean — 0.08 10% — 0.01 25% — 0.02 50% — 0.04 75% — 0.08 90% — 0.19 95% — 0.30 (RfD) 99% — 0.71
Specific population(s) at increased risk	Hg^0- Latin American and Caribbean immigrants and those of Latin American and Caribbean descent. Those with large numbers of dental amalgams. Hg^{++} - Consumers of private well water drawn from the Kirkwood-Cohansey aquifer, as well as possibly, other

	private well water. MeHg - children born to mothers with elevated consumption of high Hg-containing fish, and young children consuming significant quantities of fish or exposed through breastmilk consumption. (Note: African-Americans appears to be at less risk as a result of reduced intake of high Hg-containing fish).
Quantification of exposure levels to population(s) at increased risk (include indoor air as separate category as appropriate)	Hg^0 - Unknown for those with exposure to Hg^0 cultural/folk practices. For those exposed from dental amalgams, exposure in the upper range of intake reported by WHO (1991)(21 : g/day), the intake of Hg^0 is approximately 3.5 times the RfC (RfC = 0.3 : g/m^3. Assuming the standard default breathing rate of 20 m^3, exposure at the RfC corresponds to 6 : g/day). Hg^{++} - >1,000 persons in NJ exposed to levels above the MCL in drinking water (2 μg/l), mean Hg concentration for those exceeding the MCL was 8 μg/l, and maximum observed concentration was 36 μg/l [2] MeHg - women of childbearing age - [9] percentile of population — av. daily dose (μg/kg/day) mean 0.09 10% 0.01 25% 0.02 50% 0.05 75% 0.09 79% 0.10 - RfD 90% 0.17 95% 0.25 99% 0.43
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed	Hg^0 - RfC (inhalation) = 0.3 μg/m^3 (no estimate of risks at exposures greater than the RfC) Hg^{++}- RfD (ingestion, incl. water) = 0.3 μg/kg/day (no estimate of risks at exposures greater than the RfD) MeHg - RfD (ingestion) = 0.1 μg/kg/day Some indication of threshold for effects in range of 0.4-0.6 μg/kg/day [6].
Risk Characterization	

Risk estimate(s) by population at risk	Hg^0 - Unknown for cultural/folk practices. For those with large numbers of amalgams, the HQ (hazard quotient) may be as large as 3.5. Hg^{++} - HQ (hazard quotient)>1 for some private well consumers of Kirkwood Cohansey aquifer. Mean HQ for those >1 = 4. Maximum observed HQ = 18. MeHg - general adult population – [9] ~5% exceed the general adult RfD ~1% have a HQ >2 women of childbearing age/pregnant women - [9,10] 10-21% exceed the RfD for developmental effects ~5% have a HQ >2 ~1% has a HQ >4 1-2% have exposures in the range of a possible threshold for effects.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Hg^0 - Assuming intentional and uncontrolled residential use of Hg^0 contamination, effects may reach levels of frank symptoms. Clinical or frank effects have not been documented as a result of dental amalgam exposure. However, subtle and idiopathic symptoms may occur. Some possible symptoms may not be reversible (depending on the length of exposure). Effects on the developing fetus (maternal exposure during pregnancy) may not be reversible. Hg^{++} - at anticipated levels of residential exposure, adverse renal effects may be mild and reversible. MeHg - at currently observed levels of exposure, effects on adults and developing fetuses are likely to be subtle. Adult-type effects may be at least partly reversible. Effects due to *in utero* effects are probably not reversible.
Size of population(s) affected	Hg^0 - unknown Hg^{++} - >1,000 MeHg - unknown for general adult population. For developmental effects, 1-2% of births are in the range where subtle effects have been observed in some studies.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	Hg^0 - H - no information on size of population using Hg^0 for folk/religious practices in NJ or of resulting secondary residential air levels. Also no data on prevalence of unintentional residential releases. The extent of exposure to or the extent of subtle or idiopathic symptoms from dental amalgams is not known.

	Hg^{++} - H - limited data on size of population affected, range of exposures above the MCL, and shape of the dose-response curve. MeHg - M - Incomplete information on the shape of the dose-response curves, for adult as well as developmental effects, and lack of data on high-end fish consumers.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	Hg^0 - H - Based on very preliminary estimates from N.Y. City and CT., significant percentages of the Latin American and Caribbean populations in NJ could be found to use and be exposed to Hg^0 in residences. Hg^{++} - M - Given non-random and geographically incomplete sampling of Hg levels in groundwater, additional contaminated drinking water sources and affected populations may be identified. MeHg - L - Data on exposure levels and size population exposed in NJ is of good quality and reflects two separate and independent assessments [9,10]. Data for high-end fish consumers may show greatly elevated exposure levels, but will reflect exposure in <1% of the general population, or the population of pregnant women.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	Hg^0 - + - Reductions in the use of dental amalgams as the incidence of dental caries declines due to fluoridation and fluoride treatments. Hg^{++} - + - decrease in private well water exposure with increased installation of in-system treatment systems in affected areas. MeHg - + - reduction in consumption of high Hg fish by pregnant women with increased public awareness
Potential for catastrophic impacts (H,M,L) and brief description	L for all forms of Hg.
Extent to which threat is currently regulated	Remedial assistance is provided for private wells exceeding the MCL. Fish consumption advisories for Hg contamination are issued and distributed. Emissions limits are in place for Hg in sewage sludge for land application, stack emissions, and surface water discharge permits. Intentionally added Hg has been banned in most consumer batteries.
Relative Contributions of Sources to Risk (H,M,L)	
NJ primary sources	
Large business/industry	Hg^0 - L Hg^{++} L MeHg - M (indirectly through atmospheric emission of Hg^{++})
Small business industry	Hg^0 - L

	Hg^{++} - L MeHg - L
Transportation	Hg^{0} - L Hg^{++} L MeHg - L
Residential	Hg^{0} - H Hg^{++} L MeHg - L
Agriculture	Hg^{0} - L Hg^{++} H (historical) MeHg - M (potential runoff of Hg in agriculturally applied sewage sludge).
Recreation	Hg^{0} - L Hg^{++} L MeHg - L
Resource extraction	Hg^{0} - L Hg^{++} L MeHg - L
Government	Hg^{0} - L Hg^{++} L MeHg - L
Natural sources	Hg^{0} - L Hg^{++} L MeHg - L
Contaminated sites	Hg^{0} - M Hg^{++} M MeHg - M
Dental practices	Hg^{0} - H Hg^{++} L MeHg - L
Diffuse and non-NJ sources	
Sediment	Hg^{0} - L

	Hg^{++} L MeHg - H (particularly with disturbance and redistribution of marine and estuarine sediments)
Soil	Hg^{0} - L Hg^{++} H MeHg - H (long-term movement of watershed soil Hg (resulting from atmospheric deposition) into aquatic systems)
Non-local air sources (including deposition)	Hg^{0} - L Hg^{++} L MeHg - H
Biota sinks	Hg^{0} - L Hg^{++} H MeHg - H

Issue: Mercury
Author: Alan Stern
Version: 12/18/00

Human Health Issue Summary: Mercury

What is it?
Mercury is a naturally occurring element that has been used in a variety of industrial and commercial applications. The primary source of mercury in the environment is air deposition—quantities of mercury are released from waste incinerators, manufacturing processes, and as a byproduct of coal-burning power plants. Mercury emissions may travel hundreds of miles before precipitating out of the atmosphere, thus a portion of New Jersey's mercury deposition originates out of state. In aquatic environments, deposited mercury will react with bacteria to form methyl mercury, an organic form that accumulates in biological (e.g., fish) tissue. It is this organic form of mercury that presents the greatest human and ecological hazards.

What's at risk?
Unborn babies whose mothers consume mercury-contaminated fish during pregnancy are at risk for developmental effects. A small number of private wells may also contain unsafe concentrations of mercury. Individuals with large numbers of dental fillings may be at increased risk, as are people who intentionally use mercury in their homes for religious reasons.

What are the human health impacts in New Jersey?
A child exposed to methyl mercury *in utero* may exhibit subtle learning deficits. About 10-20% of pregnant women who consume fish may expose their children to unsafe levels; thus an estimated 11,000-24,000 infants may be exposed each year. Health effects from low-level exposure in adults are relatively mild (e.g., tremors) and reversible compared with the effects on the developing fetus. Of 2,239 private wells tested in Ocean and Atlantic counties, 59% had detectable levels of mercury, and 14% had levels that exceeded the Maximum Contaminant Level (MCL). These percentages, however, cannot be generalized to New Jersey or to Ocean and Atlantic County residents. There are no reliable estimates of the numbers of people using mercury intentionally, or how many may be at greater risk as a result of dental work.

What's being done?
Fish consumption advisories are intended to limit consumption of mercury-contaminated fish, and increased education and public awareness should help reduce human health risks. Mercury in consumer products has largely been banned or voluntarily reduced. Controls on emissions further reduce atmospheric concentrations. Assistance is provided for households with private wells exceeding the MCL.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)

Hg^0 - M - 3	Hg^0 residential - L -2 Hg^0 dental - H - 4	Hg^0 - ? (extent of residential exposure to Hg^0 due to practices specific to certain ethnic groups is not known	Hg^0 - M - 3
Hg^{++} - L - 2	Hg^{++} - M - 3	Hg^{++} - Y (areas of S. NJ with common groundwater sources)	Hg^{++} - L/M - 2
MeHg - M -3	MeHg - H - 4	MeHg	MeHg - M - 3
			M - 3

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Issue: Methyl tertiary butyl ether (MTBE)
Author: Gloria Post
Version: 05/22/02

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Methyl tertiary butyl ether (MTBE)**
Description of stressor (including etiology)	Methyl tertiary butyl ether (MTBE) is a synthetic chemical which is added to gasoline as a fuel oxygenate. It has been used in gasoline since 1979 as an octane enhancer to replace lead. More recently, it has been used to reduce air levels of carbon monoxide and ozone resulting from automobiles, under the Clean Air Act. MTBE migrates through soil more rapidly than other components of gasoline such as benzene, toluene, ethybenzene, and xylene. Therefore, groundwater contamination by MTBE after a spill or leak of gasoline from an underground storage tank often occurs prior to contamination by other gasoline components.
Stressor-specific impacts considered including key impacts	Acute: Inhalation of high levels (above those encountered in the environment) causes CNS depression. Anecdotal reports of symptoms such as headache, eye irritation, dizziness are not substantiated in general by controlled exposures. Chronic and sub-chronic: Oral and inhalation exposures of animals to high levels causes kidney effects. Weight of evidence for carcinogenicity warrants classification as Group C (possible human carcinogen). Taste and odor: complaints from water containing MTBE at levels below those of health concern.
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	Inhalation from use of oxygenated gasoline (e.g., during refueling). Ingestion of contaminated drinking water. Inhalation during household use of contaminated water (e.g., showering).
Population(s)/ecosystem(s) exposed statewide	The general public is exposed via inhalation when fueling vehicles with gasoline containing MTBE. Individuals working at gasoline stations and garages would have higher exposures via inhalation.

Do these public water supplies include surface water, or just groundwater? If just groundwater, that should be noted. I am checking this.	MTBE has been detected in public water supplies and private wells, resulting in exposure via ingestion by individuals using these water sources. MTBE has also been detected in lakes in which gasoline powered boats are used, so exposure when swimming in these lakes is possible.
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	In ambient air monitoring done by NJDEP in Camden as part of the Urban Air Toxics Program, MTBE was detected in 29 of 31 samples with a range of 0.28 to 3.74 ppb by volume and a mean of 1.29 ppb by volume. During a study of personal exposure during refueling of automobiles, the mean concentrations exceeded 300 ppb with a peak of 4100 ppb. Concentrations measured in the automobile cabin during commuting averaged 7 ppb (Lioy et al., 1994). In monitoring of public water supplies from 7/97 to 9/98, MTBE was detected at 0.5 μg/L or above in 15% of community water systems and 16% of nontransient noncommunity water systems. The highest level found in a community water system was 8.4 μg/L. Only two of 397 nontransient noncommunity systems exceeded 20 μg/L, and only one exceeded the MCL of 70 μg/L. Much of the information on private wells comes from situations in which wells were tested because of suspected contamination. There is only limited information on the occurrence of MTBE in randomly selected private wells in NJ. MTBE has been detected in wells near gasoline spills at concentrations up to several hundred parts per billion. MTBE has been found in lakes where gasoline powered boats are used at concentrations up to 29 μg/L.
Specific population(s) at increased risk	Some individuals have reported acute symptoms from inhalation of MTBE in gasoline. These effects have not been conclusively confirmed in controlled experiments. There are no known susceptible populations for effects resulting from MTBE in water.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	Exposure levels to people who have reported sensitivity would be the same as the general population.
Dose/Impact-Response Assessment Quantitative dose/impact-assessment employed for each population considered	Inhalation USEPA has not classified MTBE as to carcinogenicity. The Reference Concentration for chronic inhalation exposure on the IRIS data base is 3 mg/m^3 (0.83 ppm) based on increased kidney weights, increased severity of renal lesions, increased prostration, and swollen periocular tissues in a chronic rat inhalation study (Chun et al., 1992).
	Oral MTBE has been classified as a possible human carcinogen for oral exposure by NJDEP (group C). The oral Reference Dose developed by NJDEP is 0.01 mg/kg/day, which includes an additional 10-fold uncertainty factor for possible carcinogenicity. The Reference Dose is based on increased kidney weight in a subchronic oral rat study (Robinson et

	al., 1990).
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	Reported air levels do not approach the chronic reference dose, except for those encountered during refueling, which has a duration of only about 5 minutes or less. Individuals with private wells exceeding the MCL are at risk for excessive ingestion exposure. The number of such private wells is unknown. The taste and odor threshold for MTBE has been reported to be below the MCL of 70 µg/L.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Inhalation Acute effects from inhalation such as nausea, headaches, etc. are reversible. Chronic effects such as kidney damage and possibly cancer are severe and potentially irreversible, but are not likely to be relevant to exposure levels encountered in New Jersey. Ingestion Chronic effects such as kidney damage and possibly cancer are severe and potentially irreversible, but are not likely to be relevant to exposure levels encountered in New Jersey.
Size of population(s) affected	All drivers in New Jersey likely to be exposed via inhalation. The actual number of people exposed via drinking water is unknown.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	Oral carcinogenicity -H-The data from the only chronic oral study available was not reported in the standard fashion. It would be desirable for the NTP to conduct an oral study on MTBE. Private well exposures -H -The prevalence in private wells of MTBE at concentrations exceeding the MCL is not known.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	H -If the information mentioned above is obtained.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, -, =, □ where + is improvement)	++ Due to increased awareness of MTBE contamination of water, federal actions are underway to eliminate/reduce MTBE in gasoline. However, MTBE degrades very slowly in the environment so MTBE from existing gasoline spills is expected to persist for years in soil and groundwater.
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L -MTBE is not expected to result in catastrophic effects.
Extent to which risks are currently reduced through in-place regulations and controls	New Jersey has a drinking water standard (MCL) for MTBE, and monitoring by all public water supplies is required. The MCL also forms the basis for the groundwater cleanup criterion and the surface water criteria for MTBE. The same health basis will be used for the soil cleanup standards, when proposed.
Relative Contributions of Sources to Risk (H,M,L)	

Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	L
Small business industry	L
Transportation	H -gasoline used for autos is main source
Residential	M -fuel spills of gasoline for lawn mowers, etc. on the ground
Agriculture	M -fuel spills from tractors, etc. on ground
Recreation	M -Gasoline from two cycle engines for boats, jetskis, etc. can contaminate lakes.
Resource extraction	L
Government	L
Natural sources	L
Contaminated sites	H -Leaking Underground Storage Tanks
Diffuse and non-NJ sources	
Sediment	L
Soil	L
Non-local air sources (including deposition)	Low levels in shallow groundwater (<1 ppb) are thought to result from precipitation from ambient air.
Biota sinks	L

Human Health Issue Summary: MTBE

What is it?
Methyl tertiary butyl ether, or MTBE is a fuel additive that reduces the generation of carbon monoxide and ozone-forming compounds when burned in automobiles. The chemical is water-soluble, and when spilled migrates readily through soil and into ground water supplies. Inhalation of high concentrations of MTBE can cause nervous system depression, and animal studies have shown long term exposure to result in kidney toxicity.
What's at risk?
MTBE can be inhaled during automobile refueling and ingested via contaminated drinking water. Therefore, the entire population is generally exposed, with some increased risks

Issue: Methyl tertiary butyl ether (MTBE)
Author: Gloria Post
Version: 05/22/02
for those relying on well water that could potentially be contaminated with MTBE. Service station attendants are also at an increased risk of exposure.

What are the human health impacts in New Jersey?
Personal exposures, such as during refueling at service stations, can exceed the reference dose, but ambient concentrations are several hundred-fold lower. There are anecdotal reports of individuals suffering from acute symptoms, including headache, eye irritation, and dizziness. There are several wells contaminated with MTBE in New Jersey, but only one public water supply has exceeded regulatory levels. Contamination of private wells occasionally results in MTBE levels that exceed the maximum contaminant level (MCL) as set by the state.

What's being done?
The use of MTBE is being phased out to reduce its negative environmental impacts, particularly well contamination.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discreter communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
Acute Inhalation Effects -2, L	2, L	2, Maybe	2, L
Chronic Inhalation Effects -1, L	1,L	1, N	1, L
Chronic Oral Effects - 2, L	1,L	2, Y -If private wells in that community have elevated levels.	1, L
			1, L

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Nickel**
Description of stressor (including etiology)	Nickel is a silvery-white metal that is very abundant in nature. It can be detected in all parts of the environment including plants and animals used for human consumption, air, drinking water, rivers and lakes. It is usually found in nature as a component in various types of ores. Nickel is combined with other metals to form alloys that are used in many consumer products (i.e., kitchen utensils, jewelry, coins, pipes and faucets). Soluble nickel compounds are used in or can be generated from industrial processes (i.e., electroplating, textile dyes, and catalysts). Nickel carbonyl can be found as both a gas and liquid. Nickel carbonyl is the only gaseous nickel compound of environmental importance which may be formed inadvertently in various industrial processes that use nickel catalysts, such as coal gasification, and petroleum refining. (4) Gaseous nickel carbonyl, under ambient conditions in moist air decomposes to form nickel carbonate. Thus, in the atmosphere at concentrations near the ppb level, it has a half-life of about 30 minutes. (4) Nickel carbonyl is also a colorless to brownish liquid that is used in refining nickel ore, forming nickel films and is used as a catalyst in chemical reactions. (2) Nickel subsulfide is a major component of refinery dust and shown to produce the highest incidence of tumors for nickel compounds in animals. (8)
Stressor-specific impacts considered including key	Very small amounts of nickel have been shown to be essential for normal growth and reproduction in some species of animals, and may also be essential for humans. A daily requirement of 50 micrograms (ug) has been estimated for

impacts	humans. (5). Given the likely human dietary requirement for nickel and the lack of widespread occurrence of elevated levels of nickel in food (relative to the dietary requirement), dietary exposure is not evaluated here as a significant impact. Skin allergies, namely, contact dermatitis, is the most common effect of both occupational and non-occupational exposure to nickel alloys and soluble salts. Symptoms can include a skin rash (dryness and itching) on the fingers, wrists and forearms. (5) Laboratory animals were observed to have decreased body and organ weight. (8) Chronic (long-term) respiratory effects such as asthma and an increased risk of chronic respiratory tract infections in humans have been associated with exposure to nickel. Nickel subsulfide is listed as a Group A carcinogen producing lung and nasal cancer. (8) Nickel carbonyl is listed as a Group B2 carcinogen producing lung and nasal cancer (8) and was also found to be a teratogen in animals. (2)
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	Exposure to nickel can result from daily contact with consumer products through dermal and ingestion. Contributions of atmospheric nickel come from both natural sources and anthropogenic activity. (4) Various dry and wet precipitation processes remove particulates from the atmosphere and may be transferred to soil and water. Inhalation: Ambient air contains low concentrations of nickel resulting from manufacturing facilities, oil and coal combustion, sewage sludge incineration and other sources. (5) Ambient air concentration in US cities ranges from 5 to 50 ng/m^3. Smoking tobacco is another major source of nickel exposure. Nickel has been implicated in lung cancer due to occupational exposure in nickel refineries. However, there are no active nickel refineries in the US, therefore there is little exposure to refinery dust and nickel subsulfide. (7) Oral: Food is the major source of nickel ingestion. The average daily intake for adults is 100 to 300 ug per day. (5) Drinking water is also another potential source of nickel ingestion. Uptake of nickel from drinking water averages 2 ug/day and 170 ug/day from food. (11) Dermal: The general population is exposed daily to consumer products made of or containing nickel. Nickel doesn't readily penetrate skin. Individuals with skin allergies to nickel consist of 2.5 to 5 percent of the population. (7)
Population(s)/ecosystem(s) exposed statewide	

	Because of the ubiquitous nature of nickel and its use in everyday household items, the statewide population is exposed on a daily basis through inhalation, ingestion and dermal contact. The primary exposure route is ingestion.
Quantification of exposure levels statewide (include indoor air as separate category as appropriate)	Query results (6) for nickel contamination in drinking water systems for the State of New Jersey indicated that nickel was not detected in concentration that exceeded the maximum contaminant level. Air emission data (13) indicates that 21.5 tons of nickel compounds per year are emitted. The highest emissions were from nonferrous foundries and industrial boilers (residual oil). Modeled concentrations (14) for nickel (mean) in air for New Jersey counties ranged from 1.8 to 49.8 ng/m^3.
Specific population(s) at increased risk	Smokers and those individuals occupationally exposed to nickel or living in the vicinity of facilities with air or surface water emissions of nickel. Individuals who may be sensitive to dermal contact with nickel (2.5 to 5 percent of the population).
Quantification of exposure levels to population(s) at increased risk (due to factors other than exposure) (include indoor air as separate category as appropriate)	Difficult to determine however, the general risk characterization is presented below.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	Nickel soluble salts B RfD: 0.02 mg/kg/day Nickel subsulfide B CSF inhalation: 0.00048 (mg/m3)-1; CSF oral: 1.7 (mg/kg/day)-1 No RFC for nickel
Risk Characterization	

Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	Inhalation: based on the total air release for nickel in New Jersey, the following risks were provided for nickel (10). Added Cancer Risk (per 1,000,000) = 5.1 Noncancer Hazard Index = 0.39 These risks were calculated using the EPA generic exposure assumptions of adults (70 kg) breathing 20 cubic meters air per day over a lifetime (70 years) exposure and absorbing 100 percent of the nickel present in air. No information was presented on the form of nickel that was present. Air concentrations for NJ counties were found to be similar to ranges found across the United States. Risk was not calculated since the speciation of nickel compound present was not known. Smoking was not evaluated since it is a voluntary act. Ingestion: Drinking water may not be a potential concern based on query, but does not include private wells. Dietary was not evaluated. Dermal: Sensitivity of the New Jersey population unknown. It may be reasonable to assume that 2.5 to 5 percent of the population may experience allergic dermal effects, with a smaller percentage experiencing allergic effects.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Inhalation: Some asthmatic symptoms are reversible. Resulting cancer is considered severe. Ingestion: Noncancer endpoints (increased urinary albumin levels, reduced body weight and organ weight) considered potentially persistent to reversible. (11) Dermal: Contact dermatitis considered mild and reversible. However, if sensitized to nickel, complete recovery may be difficult.
Size of population(s) affected	Because of the ubiquitous nature of nickel and its use in everyday household items and consumer products, the statewide population is exposed on a daily basis. Populations at an increased risk resulting from air emissions is unknown.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	Medium B Use of EPA database for quantitative emissions data and generalization of data to entire state. Cumulative emissions likely increase the overall cancer/noncancer risk estimates. Medium B Releases to water may be underestimated if nickel evaluation is not part of the routine monitoring. High B The fraction of the population experiencing nickel allergic contact dermatitis is unknown.

Potential for future changes in the underlying risk from this stressor,	Low
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	Low
Extent to which risks are currently reduced through in-place regulations and controls	Drinking water MCL has been established at 0.1 mg/l. Current air and water pollution control regulations. The NJDEP Residential Soil Cleanup Criteria for nickel - 250 mg/kg. OSHA PEL and ACGIH TLV (5): Metal and insoluble nickel B 1 mg/m3 Soluble inorganic nickel B 0.1 mg/m3 Nickel Carbonyl - 0.007 mg/m3
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	Medium B atmospheric emissions and water discharge
Small business industry	Medium B atmospheric emissions and water discharge
Transportation	Low B minor source of atmospheric nickel from combustion of gas and diesel fuel (1)
Residential	Low B Plumbing and faucets that contain nickel may significantly increase nickel in drinking water (7). Fuel combustion (ex. residential oil, coal) is responsible for the majority of total statewide emissions of nickel. (12)

Agriculture	Low B Nickel in diet can be increased by the use of certain fertilizers on food crops (7).
Recreation	Low
Resource extraction	Low B US contributed 0.7 percent of nickel mined in the world. (1) Mining not occurring in New Jersey.
Government	Low
Natural sources	Low B Nickel's abundance in the earth's crust is 0.018 percent and is found in many ores such as sulfides, arsenides, and oxides or silicates. Natural sources of airborne particles that contain nickel include soil, sea spray, volcanoes, forest fires, and vegetation. Wind erosion and volcanic activity contribute 40-50 percent of the atmospheric nickel from natural sources. (12)
Contaminated sites	Sites listed as having nickel, but no information available to determine if nickel was elevated above background concentrations.
Diffuse and non-NJ sources	
Sediment	Nickel will bind to sediment. Potential for elevated concentrations above background is medium. Availability will determine whether there is a potential concern. Nickel has been identified as a parameter of concern in surface water for the NY-NJ Harbor. Nickel concentrations (mean concentration of 48.9 mg/kg) in sediment exceeded sediment screening concentrations based on ecological endpoints. However these concentrations are below the residential soil cleanup criteria. Potential for these concentrations to be a concern for direct contact is low.
	Nickel is a naturally occurring metal. Deposition of air emission may result in concentrations exceeding natural background. Typical range for New Jersey soil is 5 to 25 mg/kg. More developed areas (i.e., northern Jersey) soil concentrations are slightly higher with the low end of range being 15 mg/kg.

Non-local air sources (including deposition)	Medium
Biota sinks	Low B nickel doesn't bioconcentrate in plants, fish or animals.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
Low	Low	Low	Low

References:

(1) Agency for Toxic Substance and Disease Registry (ATSDR) Toxicological Profile for Nickel.

(2) New Jersey Department of Health and Senior Services, 1994. Hazardous Substance Fact Sheet: Nickel Carbonyl.

(3) ATSDR, 1997. ToxFAQs Nickel. www.atsdr.cdc.gov/tfactsis.html

(4) USEPA, Drinking Water and Health Technical Factsheet on Nickel. www.epa.gov/ogwdw/dwb/tioc/nickel.html

(5) USEPA, Nickel and Compounds. www.epa.gov/ttn/uatw/hlthef/nickel.html

(6) National Contaminant Occurrence Query for Drinking Water Data. www.epa.gov:9966/ncod/pk_ncod_query_html.

(7) ATSDR, 1988. Public Health Statement: Nickel. www.atsdr.cdc.gov/toxprofiles/phs8819.html

(8) Integrated Risk Information System (IRIS). EPA online database. www.gov/iris/index.html

(9) Scorecard. About chemicals/by state. www.scorecard.org/env-release/

(10) Scorecard. Pollution locator/hazardous air pollutants/chemical-specific emission sources for Jersey. www.scorecard.org

(11) Toxicological Excellence for Risk Assessment, 1998. Toxicological Review of Soluble Nickel Salts. External review draft. July 28, 1998.

(12) Scorecard. Nickel and Compounds. www.scorecard.org/chemical-profiles/html/nickel.html

(13) New Jersey Department of Environmental Resources 1996. Nickel Emissions for 1996 NTI Area Sources.

(14) EPA 1990. Cumulative Exposure Project. 1990 Modeled Nickel concentrations.

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Nitrogen Pollution (Water)**
Description of stressor (including etiology)	Nitrate ion refers to the anionic inorganic species containing a nitrogen atom and three oxygen atoms, which carries a single negative charge in water (NO_3^-). It is most often associated with other positively charged inorganic ionic species. Most common forms of inorganic nitrate are highly soluble in water. Nitrate can enter both groundwater and surface water through run-off and infiltration into shallow aquifers from past and present agricultural fertilizer application, sewage and animal waste contamination, and through the deposition of nitrate-containing aerosols derived from fossil fuel combustion.
Stressor-specific impacts considered including key impacts	There has been a great deal of study on the effects of nitrate in drinking water. Generally speaking, the major health impact resulting from the ingestion of high levels of nitrates in drinking water are related to the development of methemoglobinemia, which is often referred to as "blue-baby syndrome" when it occurs in infants less than 6 months old. This condition involves a potentially serious reduction in the ability of the blood to carry oxygen throughout the body and results from the oxidation of the ferrous ion in hemoglobin to the ferric ion by nitrite, creating what is called methemoglobin. Nitrates are reduced in saliva and to some extent in the gastrointestinal tract to nitrite, which enters the blood and reacts readily with hemoglobin. Methemoglobin does not bind oxygen and thus does not carry oxygen throughout the body. Methemoglobin levels greater than 10% of total hemoglobin can result in clinical anoxia while levels of 60% or greater can result in death. Methemoglobinemia from nitrates, although infrequent in the United States, is a phenomenon primarily observed in infants under the age of 6 months because of their unique susceptibility. Over the last several decades some deaths have been reported and attributed to nitrate in drinking water. Infants consume more liquid per unit body weight relative to adults. Infants under the age of 6 months are deficient in an important enzyme, methemoglobin reductase, which is responsible for catalyzing the reduction of methemoglobin back to hemoglobin. This is an important defense mechanism that prevents levels of methemoglobin from accumulating in the body of an adult. Infants are also more susceptible because they have fetal hemoglobin (hemoglobin F) which is more easily oxidized to methemoglobin compared to adult hemoglobin (hemoglobin A), but by 6 months of age there are dramatic increases in hemoglobin A in infants. Infants have a limited capacity to produce gastric acids and thus may have a stomach pH between 5 - 7. Higher pH levels allow for the colonization of the digestive tract by micro-organisms that are able to reduce nitrate to nitrite. This mechanism may also explain the association of methemoglobinemia with bacterial infections of the gastrointestinal tract.

	A small number of persons have a hereditary deficiency in methemoglobin reductase. Persons of Navajo Indian, Alaskan Eskimo and Indian, and Puerto Rican descent have a higher prevalence rate for this genetic condition (an autosomal recessive trait). In addition, persons with other enzyme deficiencies or pre-existing conditions and full-term pregnant women are more susceptible to methemoglobinemia. Pregnant women are most susceptible at or near the 30th week of pregnancy. The current MCL of 10 mg/L for nitrates in drinking water was derived from studies of methemoglobinemia. It has been shown that there have not been any cases of methemoglobinemia that have resulted from children and infants consuming water that had less than 10 mg/L of nitrate nitrogen. Typically most cases of methemoglobinemia have resulted from concentrations greater than 50 mg/L of nitrate nitrogen in drinking water, but there was a very small percentage of cases that have occurred at levels between 10 - 20 mg/L. Some of these cases at lower levels were also accompanied by concurrent gastrointestinal illness, which may have greatly increased the infant's susceptibility. In recent years, some attention has also been given to the potential for nitrates in drinking water to contribute to cancer risk and poor reproductive outcomes. These studies have speculated that nitrates and nitrites can undergo nitrosation with amines and other protein components within the gastrointestinal tract to form carcinogenic n-nitroso compounds. However, the epidemiological studies of nitrates in drinking water have had mixed results for bladder cancer, gastric cancer, and non-Hodgkins lymphoma. Some of the major challenges in interpreting these studies concerns many of the typical limitations of ecological or case-control epidemiological studies, including exposure misclassification bias and confounding. Most epidemiological studies investigating nitrates did not measure other common contaminants in drinking water. As a result, these epidemiological studies were not able to ascertain potential contribution or confounding from other pollutants, such as pesticides. In addition, dietary contributions to the total body burden of nitrates/nitrites were not accounted by these epidemiological studies. These same limitations apply to the human health studies of adverse reproductive effects. Many toxicology studies using various animal species have also been conducted to evaluate the safety of nitrate and nitrites in drinking water. Most animal studies have found no significant evidence or found equivocal evidence for the carcinogenic activity of inorganic nitrates and nitrites (principally sodium nitrate or nitrite). In a two-year chronic NTP study, female rats exposed to high concentrations in drinking water (>750 ppm) had higher rates of fibroadenoma in the mammary gland compared to controls, but male and female rats had lower rates of mononuclear cell leukemia than controls. Female mice had a positive trend in the incidence of forestomach squamous cell papillomas that had marginal statistical significance and may or may not have been chemically related. The relationship of stomach papillomas to cancer elsewhere in the body is unclear. At extremely high concentrations (3000 mg/L) there was some pathological evidence of stomach epithelial cell hyperplasia in male and female rats and male mice. Most controlled laboratory animal studies have had mixed results concerning risk of abortion during pregnancy or adverse reproductive outcomes, with typical results showing effects at very high concentrations (e.g. 30,000 ppm). Several animal studies have not demonstrated any adverse reproductive effects, despite the fact that there is some anecdotal evidence in the veterinary literature concerning increases in abortions among livestock drinking water with high levels of nitrates. There is little evidence to suggest that nitrate in drinking water has teratogenic properties. Animal studies of nursing litters have provided little evidence that mothers consuming high levels of nitrates pass nitrate on to their litters through nursing.
Exposure Assessment	
Exposure routes and pathways considered **(include indoor air as appropriate)**	This assessment only considers exposure to nitrates through drinking water. Baby formula prepared with water contaminated by nitrates is the important exposure to consider. Other significant exposure routes also exist, including consumption of certain vegetables and foods with nitrite preservatives, but these sources are not significant for infants.

Population(s)/ecosystem(s) exposed statewide	Community water systems (CWS) are public water systems that serve year round residents. Noncommunity water systems (NCWS) are public water systems that serve a nonresidential population, such as factories, campgrounds, nursing homes, schools, various retail shops, etc. Public water systems have been required to sample at least annually since 1993, and approximately once every three years going back to the 1970s. This data is submitted to the Bureau of Safe Drinking Water. Generally speaking, when problems have occurred in New Jersey they have only been found in NCWS. It can be inferred, from the NCWS data and from agricultural monitoring well data collected by the USGS, that some private wells are more susceptible to nitrate contamination and hence potentially have higher levels compared to public water systems. Proximity of wells or source waters to septic systems, fertilized agricultural lands, sewage or sludge disposal, and livestock waste is usually the source of nitrate in water.
Quantification of exposure levels statewide, including populations at significantly increased exposure **(include indoor air as separate category as appropriate)**	According to the Bureau of Safe Drinking Water (BSDW) data on nitrates for 1998 and 1999, no community water systems (CWS) had an MCL Violation for nitrate, although a handful of CWSs reported short term unconfirmed exceedences (in all situations, check samples showed the concentrations to be below 10 mg/L). All three of the unconfirmed exceedences in 1998 were less than 12.5 mg/l. Two of the three systems serve less than 100 people and one of the CWS serves 32,000, however this larger CWS obtains water from a total of 12 sources, only one of which had the one-time unconfirmed exceedence. It is unlikely that all persons served by the large CWS would have received water with short-term levels greater than the MCL during the one-time unconfirmed exceedence since there were a total of 12 wells used but only 1 well was affected during the exceedence. In 1999, there was only one unconfirmed exceedence from a CWS (at a concentration of 11.2 mg/L). This CWS serves a population of 225. Therefore, in any given year it can be roughly estimated that there may be 100 – 10,000 people (rough estimate of possible worst case) exposed to values greater than the MCL for a short period of time, but none >20 mg/L of nitrate nitrogen in CWS. As a result, CWS are expected to pose only a negligible risk for the majority of the population. Non-community water systems (NCWS) are also evaluated for nitrate concentrations even though infants are unlikely to be affected by these systems, unless the system is a daycare or nursery. In fact, at the discretion of the State, nitrate levels not to exceed 20 mg/L may be allowed in a noncommunity water systems if it is demonstrated that such water will not be available to children under 6 months of age and appropriate public notification is performed. NCWS had much greater variability in the populations served and concentrations measured. In 1998, there were 17 NCWS that had an MCL Violation for nitrate. All concentrations were 33 ppm or less. In 1999, there were 26 NCWS with an MCL Violation for nitrate, with 34 ppm being the highest measured value.
Specific population(s) at increased risk	The most vulnerable population is infants under the age of 6 months. In 1998, there were 113,850 births in the state of New Jersey. There are also people with hereditary or pre-existing health conditions that are more susceptible to methemoglobinemia relative to the normal adult, but this group is expected to be very small in relation to the overall New Jersey population. Pregnant women near full term may also be more susceptible to nitrate methemoglobinemia.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) **(include indoor air as separate category as appropriate)**	Generally speaking most drinking water in the state is within the acceptable MCL of 10 mg/L. The values measured were <15 mg/L of nitrate nitrogen in a few cases where there have been possible short term (unconfirmed) exceedences in CWS, while for NCWS they were still below 34 mg/L, which is still below the level where most cases of methemoglobinemia have been observed (50 mg/L).

Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	The data that formed the basis for setting the standard at 10 mg/L was based on public health data showing that cases of infant methemoglobinemia have not been known to occur clinically at values below 10 mg/L. Most cases typically occurred at relatively high nitrate concentrations, but there have been a few infant cases reported in the literature that resulted from the consumption of water between 10 – 20 mg/L. However, these rare cases occurred when water nitrate consumption was accompanied by gastrointestinal tract infections.
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	No cases of methemoglobinemia would be expected at or below the MCL for nitrates in drinking water. Minimal risk is expected up to 20 mg/L. Significant risk usually occurs at values closer to 50 mg/L.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate) Size of population(s) affected	Methemoglobinemia is a reversible condition that generally responds well to medical treatment. However, if very high levels of methemoglobin are left unrecognized and therefore untreated, it can result in severe anoxia and lead to death. Some infant deaths have been reported in the literature, although not in recent years. There were approximately 1,175 people in 1998 and 225 people in 1999 on CWS that may have received water greater than 10 mg/L nitrate nitrogen for a short period of time (check samples showed concentrations less than 10 mg/L), but even those levels in CWS were below 20 mg/L nitrate nitrogen. If we assume that a significant risk does not occur until values reach 20 mg/L, then CWS would not have any children at significant risk of methemoglobinemia.
	However, some private wells would be more likely to have higher concentrations of nitrate. The likelihood of nitrate contamination in private wells was derived by using NCWS as a surrogate. If it is assumed that NCWS are representative of private wells, then we can estimate the number of infants that would receive water with nitrate nitrogen values above the MCL. There were a total of approximately 3,800 NCWS in NJ in 1998 and 1999 of which 17 had an MCL Violation for nitrate in 1998 and 26 had an MCL Violation for nitrate in 1999, for an average of 22 NCWS with an MCL Violation for nitrate over the two years. This yields an average of 0.6% (=22/3800) of all NCWS in any given year with an MCL Violation for nitrate. The

	analytical results related to these violations show that concentrations were above 20 mg/L nitrate nitrogen in only 3 NCWS in 1998 and 6 NCWS in 1999 (for an average of 5 NCWS). Approximately 0.13% (=5/3800) of NCWS exceed 20 mg/L nitrate nitrogen. Therefore, of the estimated 300,000 – 400,000 private wells in NJ, we can calculate the number of private wells likely to exceed the standard by using the NCWS data as a representative surrogate: 0.6 % of the total number of private wells yields an estimate of 1,800 to 2,400 private wells exceeding the nitrate MCL and 0.13% of the total number of private wells suggests that 390 - 520 private wells are above 20 mg/L nitrate nitrogen. Assuming 3 persons per private well would yield an estimate of 5,400 – 7,200 people drinking water above the nitrate MCL and 1,170 – 1,560 people drinking water greater than 20 mg/L nitrate nitrogen. If we estimate 1.2 % of the population is less than 6 months of age (1998 births/total 1998 population), this would yield 60 – 90 infants exposed to water above the MCL, of which 10 - 20 infants would be exposed to values greater than 20 mg/L nitrate nitrogen. Thus, anywhere from 10 - 20 infants would be at risk of methemoglobinemia annually in NJ. However, the actual number of clinical cases would be smaller because many infants are breast fed and therefore do not consume tap water in the first few months of life. We can estimate the overall risk by using the 1998 birth rate of 113,850 births per year and the upper estimate of methemoglobinemia (20 children). This would yield an estimated statewide risk of infant methemoglobinemia as 2X 10^{-4}. It should be noted that there have not been any clinical reports of infant methemoglobinemia cases attributed to NJ drinking water in recent years.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	There is a significant amount of data and clinical experience with methemoglobinemia.(L) However, there are large data gaps and a high degree of uncertainty in terms of cancer and reproductive end points. (H)
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	Reducing uncertainties in terms of cancer and reproductive endpoints could significantly impact the risk characterization for this material in drinking water.(H) Methemoglobinemia (L)
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, -, =, □ where + is improvement)	++ (due to recently enacted well testing legislation in NJ)
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	There does not seem to be a high degree of probability for catastrophic events concerning nitrate in drinking water. However, intentional or unintentional (e.g., dumping fertilizer into a public drinking water distribution system) poisoning of infants and other susceptible populations could be a possibility. One instance during the 1990s in New Jersey occurred when nitrous acid being used to clean a boiler without backflow prevention resulted in high levels of nitrites entering the water system of a local school, causing children consuming water and soup in the cafeteria to experience methemoglobinemia. (L)
Extent to which risks are currently reduced through in-place regulations and controls	Risks of methemoglobinemia in the general population are reduced significantly by adhering to the current MCL.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	

Large business/industry	N
Small business industry	N
Transportation	N
Residential	Y/N
Agriculture	Y
Recreation	N
Resource extraction	N
Government	N
Natural sources	Y/N
Contaminated sites	N
Diffuse and non-NJ sources	
Sediment	N
Soil	Y
Non-local air sources (including deposition)	N
Biota sinks	N

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	**Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)**	**Are there discreter communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)**	**Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)**
Non-cancer only: 2 (M)	1 (L)	3 (Y)	2 (L)

			2 (L)

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Assessment

Risk Assessment Framework	Findings
Hazard Identification	**Nitrogen Oxides (NOx)**
Stressor	
Description of stressor (including etiology)	Oxides of Nitrogen are by products of combustion. The compound is formed under conditions of high temperature. While there are several oxides of nitrogen, nitrogen dioxide is the principle compound of interest because it is the longest lasting and most prevalent of the family and is a product that results when the less stable NO is further oxidized under atmospheric conditions. For this reason, this analysis focuses on the properties of NO_2. In addition to sources contributing to outdoor pollution, NO_2 is a significant indoor pollutant particularly in homes with gas stoves or kerosene space heaters. In these cases, indoor conditions may contribute greater exposures than outdoor conditions. NO_2 plays two roles in environmental damage. Its direct effects include damage to human health, but as a precursor to ozone, it also contributes to those effects. For this analysis, we focus on the direct impacts of NO_2. The impacts from ozone are covered in an additional analysis. (NJCRP, 2001)
stressor-specific impacts considered including key impacts	The impacts from NO_2 are primarily of the respiratory system. Secondary effects resulting from damage to the respiratory system include weakening the immune system and cardiovascular damage. (NAPAP, 1990)
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	The sole route of exposure considered in this analysis is through inhalation.
Population(s)/ecosystem(s) exposed statewide	The total population of New Jersey is exposed to significant levels of NO_2. However, somewhat elevated concentrations exist in certain urban areas (US EPA, (AIRS)). For the purpose of this analysis, we consider two populations. A general population exposed to a low level almost continuously with occasional elevated levels. A population that is exposed to levels typical of urban environments has a somewhat higher continuous exposure and a greater frequency of elevated exposures.

Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	NO_x is a criteria pollutant as described in the Clean Air Act. As such, it has been the focus of monitoring and regulation for the past 20 years. In New Jersey, there are 7 official sites that monitor NO_x on an hourly basis. The distribution of NO_x among monitoring sites is the focus of significant modeling, but the summary of such models is that there are some local variations but not sharp peaks and valleys due to local conditions. The air quality standard established under the Clean Air Act is for a site not to exceed .053 ppm averaged over a year. None of the New Jersey monitors exceed this standard.
Specific population(s) at increased risk	Children are susceptible to NO_x and its effects on immune systems. (Lippmann, 1992) Asthmatics are also susceptible to low level exposures. (NAPAP, 1990)
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations)	Within the two exposures described in this analysis, 330,000 children less than age 3 live in New Jersey. About 75,000 of these children live in the three county urban area with elevated NO_x (US Census).
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	Most controlled studies of NO_2 impacts use short-term exposures of greater than 1ppm. Any short term exposures less than 1ppm show no clinical effects (NAPAP). At higher concentrations, there is evidence of effects on the respiratory tract, but these concentrations are not important for typical environmental exposures. At 1ppm some effects on immune system response are noted (Lippmann, 1992). In addition to the clinical studies, there are epidemiological studies that attempt to find relations between urban NO_2 and elevated incidences of respiratory illness and pulmonary function. It is these studies that form the basis of regulatory levels determined for the National Ambient Air Quality Standards. These standards set a maximum annual average of .053 ppm (NJ has its own standard at .05 ppm). The total of epidemiological studies suggests that concentrations below .05 ppm will have no effect on health conditions. However, some individual studies suggest effects in children as low as .015 ppm. The most noticeable and reproduced impacts observed at low levels are the susceptibility to respiratory disease (such as cold and flu symptoms) (Berwick et al., 1984).
Risk Characterization Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	For the general population, the average concentration of NO_2 is less than .02 ppm suggesting that there is a slight chance that children may have challenged immune systems. Considering higher values, there are less than 10% of the hours with values greater than .05 ppm. (US EPA, (AIRS)) For three urban counties with monitoring sites (Essex, Hudson and Union), the year 2000 average concentrations are between .026 and .043 ppm suggesting that effects in the most susceptible populations are possible. In addition, these urban areas show peak values of .1 ppm and levels above .05 more than 10% of the time. While these are not annual averages upon which the regulatory standards are determined, the occasional high values exceed levels shown to have no effect suggesting the possibility of effect beyond the childhood immunological impacts.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	The potential effects on children's immune systems are most noticeable in the frequency and severity of childhood colds and flu. The symptoms of runny nose, sore throat, difficulty sleeping and exhaustion are not life threatening but certainly troubling to the children and their families. It is not clear if early susceptibility to these diseases has long lasting effects with respect to adult health conditions.

	The effects on pulmonary function are also not long lasting. If these individuals are removed from air pollutants, their pulmonary functions can return to normal. However, during events of reduced pulmonary function, susceptible individuals may suffer from asthma-like symptoms. (NAPAP 22-82, 1990) The frequency of elevated concentrations of NO_2 (>.05 ppm) is on the order of dozens of events per year in the urban counties and less than 10 events per year in other areas.
Size of population(s) affected	The population of children ages 0-3 in New Jersey is approximately 330,000. The population of asthmatics in New Jersey is 250,000 assuming an overall asthma rate of 3% as suggested by national studies (American Lung Association, 2001). If we assume the estimate of asthmatics at 3% of the population, then the estimate of asthmatics in the 3-county area is 54,000.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	L-M Because of the scrutiny of standards under the Clean Air Act, the impacts from exposure to NO_x are relatively well studied. However, unlike ozone and carbon monoxide, reviews of NO_x effects offer significantly conflicting results (NAPAP, 1990). For every study suggesting low concentration effects, other similar studies show no effect. As a result of this observation the certainty of effect may be less with NO_2 than with other criteria air pollutants. And, as with most air pollutants, it is difficult to separate the affects of NO_x from those of other pollutants such as particulates and ozone, which often co-exist with NO_x. Regardless of the uncertainty, there is some possible evidence that NO_x causes health effects in exposed populations.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	L-M The potential for significant changes in risk estimates is less than with most other pollutants. Clinical studies will probably never provide irrefutable evidence for impacts in the range of exposure that New Jersey faces, and epidemiological studies will add to existing studies, therefore not reversing the conclusions that have been made to date.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , ≈, where + is improvement)	0 - NO_x is a stubborn pollutant to control. Unlike carbon monoxide, which can be controlled by more efficient burning, reducing NO_x generation requires lower combustion temperatures which decreases the overall efficiency of most combustion processes. Therefore, NO_x reduction strategies require scrubbing or catalytic conversion, both of which require greater capital investment for each combustion source. Similarly, the large role that automobile vehicles play in NO_x emissions results in the challenge in continuing per mile emission reductions to counter the increasing number of vehicle miles traveled. The result of this challenge is that NO_x emissions have remained relatively constant over the past 15 years despite significant investments in reduction strategies (NJDEP, 1998). The future holds no greater promise for reductions, and increased vehicle use and increased use of other combustion facilities will make further progress challenging.
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L - Catastrophic releases of NO_x are not likely.

Extent to which risks are currently reduced through in-place regulations and controls	NO_x is one of the criteria air pollutants with specific federal standards under the Clean Air Act. As such, most states, including New Jersey have taken significant regulatory steps to control point source emissions. Regulations on vehicle emissions at the federal level have reduced per mile emissions from vehicles.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	M-H According to Environmental Defense figures, stationary sources of NO_x represent about two thirds of the entire emissions in New Jersey. However, stationary sources include domestic boilers as well as utility and large industrial external combustion units. (Environmental Defense)
Small business industry	M
Transportation	M – Vehicle use represents the other third of emissions in New Jersey (Environmental Defense)
Residential	M-L – Small boilers are typically smaller contributors of NOx.
Agriculture	L
Recreation	L-M – Off road vehicles may be significant contributors.
Resource extraction	L
Government	L
Natural sources	L
Contaminated sites	N/A
Diffuse and non-NJ sources	
Sediment	N/A
Soil	N/A
Non-local air sources (including deposition)	M-H – NO_x is a mobile and long lasting pollutant. Evidence from ozone monitoring shows that sources up to 1,000 miles away can contribute to local NO_x concentrations. New Jersey is downwind from several large sources, but is also upwind from populations in New England (NESCAUM, 1997).

Biota sinks	

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
Asthmatics (M-L) 2-L/M	2-L/M	Slightly (urban) 2-L/M	3-M
Children (M-L) 2-L/M	2-L/M	Slightly (Urban) 2-L/M	3-M
			3-M

Human Health Issue Summary: Nitrogen pollution

What is it?
The natural nitrogen cycle is disrupted by the use of nitrogen fertilizers and by the production of nitrogen oxides during combustion. Excess nitrogen from fertilizers enters aquatic ecosystems, causing algal blooms and reducing oxygen levels and other ecological effects. Oxides of nitrogen (NO_x) are by-products of combustion that can cause damage to the respiratory and cardiovascular systems when inhaled. Oxides of nitrogen are present in precipitation, adding to the ecological impacts caused by fertilizer runoff. NO_x is a precursor to ozone and a constituent of acid precipitation; the effects of those are described in separate reports.

What's at risk?
Virtually the entire population is exposed to NO_x and residents of urban areas are exposed to somewhat higher levels. As with other air pollutants, NO_x can exist at higher concentrations indoors and pose greater risk. At particular risk are asthmatics and children.

What are the human health impacts in New Jersey?
The concentration of NO_x in New Jersey is below federal regulatory standards, but there is some evidence that the concentrations that do exist in New Jersey can increase the susceptibility of children to respiratory disease. There is some evidence of increased incidence of asthma among the approximately 54,000 asthmatics that live in the three New Jersey counties with highest ambient concentrations. For both children and asthmatics, indoor exposures increase the risk.

What's being done?
Both airborne NO_x and applied fertilizers are the focus of several programs. New Jersey is required to reduce NO_x emissions to comply with federal regulations. Fertilizer use is not regulated, but efforts to reduce the incidence of excessive use are important in watershed management efforts.

Issue: Nitrogen Oxides
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Refernece:

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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Noise Pollution**
Description of stressor (including etiology)	Noise is generically described as "unwanted sound". However, regardless of the perception of the receiver as to whether the exposure is "noise" or "sound", excessive exposure can have various physiological effects. Noise exposures among the population result from the impact of acoustic energy through air to the tympanum (ear drum). The impulses are subsequently transmitted through the inner ear and processed to nerve signals that can be perceived by the individual, creating a number of physiological and physical impacts. Assessment of noise risk and identification of mitigating factors is complicated by the fact that exposures can be voluntary or imposed; the exposed individuals may have full, limited or no control over the noise source or their personal protection; and the noise can be perceived as desirable (even sought) or undesirable. Sources can be categorized as: Environmental, from vehicle / aircraft traffic, highways, airports, railroads, or industry; Workplace related, including military; Recreational / group (sporting events / rock concerts); Recreational / personal (hunting, radio / television, headphones, instrumental music); Lifestyle / personal (homes / building interiors, vehicles).
Stressor-specific impacts considered including key impacts	Noise induced hearing loss resulting from cumulative exposure to significant / high noise doses to individuals or groups: Assessment of noise-induced hearing loss is complicated by presbycusis (hearing loss associated with the aging process). Specific cause of presbycusis is not fully understood, but it could at least partially be related to cumulative noise exposure. Sleep disturbance. Direct impact on other (non-aural) physiological systems, such as the cardiovascular system (blood pressure, heart rate). Psychological impact from sleep deprivation or other subjective responses to noise. Interference with communications, particularly telephone conversations.
	This assessment does not address pure "quality of life" issues (e.g., residence near a significant though non-damaging environmental noise source) or the psychological impact of noise that might be regarded as objectionable by some,

	but not all, among the general population. This assessment does not include the potential for a physiological response to vibration which might be transmitted to the body through contact with solid vibrating structures, or even through the air at low frequencies of high power, though they might be imperceptible to the ear. In addition, this assessment does not address accidents/ injuries that might result from distractions or noise that might interfere with speech/ instructions, alarms/ signals, or other audible warnings (horns, sirens, etc.).
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	Air only
Population(s)/ecosystem(s) exposed statewide	Entire general population. While wildlife habitats could be affected, any such impact is not considered in this assessment.
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	Several aspects of noise are not considered here: Workplace noise exposures; Personal exposures where the source of noise and protective measures are fully within the control of the individual (e.g., radios / headsets, hunting); Mini-environments, where noise sources are typically from personally-owned consumer products; or where the source and controls are at least partially within the control of the individual (e. g., interiors of personal vehicles, residential appliances / heating / ventilation, console radios / TVs); and Accident injuries that could result from interference with speech/ communication, alarms/ signals or other audible warnings.
Specific population(s) at increased risk	Removal of the above exposure sources and groups tends to largely eliminate noise-induced hearing loss as a primary effect for this assessment. Lower levels of noise ("background"/environmental) could impact what is typically considered as being presbycusis. Remaining population at risk {tc \l 1 "Remaining population at risk"} Sleep-deprived individuals, and individuals with irregular sleeping patterns, including shift workers; People with medical conditions that impact their ability to sleep, including the aging population with reduced capacity to sleep soundly; Physically or psychologically ill and / or hospitalized; Those who are not acclimatized to significant noise, e.g., new residents and infants Individuals with compromised cardiovascular systems.

Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	Exposure information needed: # of people within X kilometers of commercial airports; # of people living within Y yards (X100) of commuter or freight railroads; # of people living within Z yards (X 100) of an interstate highway. Additional (higher exposure) information on people living near toll plazas, and intersections of interstates / major highways; # of people living near major state / federal roads /highways, particularly near stoplights and significant grades (trucks); # of people living near sports venues and entertainment districts; # of people living near noise-producing industrial areas and marine / trucking terminals; and # of people living near commercial districts and in urban areas.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	Not done
Risk Characterization	
risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	Not done
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Not done
size of population(s) affected	
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	High Dose effect relationship among exposed people. Clarification of confounding factors associated with observed effects. Additional noise level data and statewide distribution.
	High

Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	See above
	+ Improvements in risk factors associated with lifestyle factors or personal choices can be mediated through public education/ training and technological improvements in some consumer products (appliances, vehicles, musical instruments, audio equipment). Urban planning controls can have moderate effect over time in currently developed areas (sound barriers, airport management, industrial settings, community noise standard). Urban policy/ planning is likely to have the most impact by mediating against future degradation of the noise environment in undeveloped or moderately developed areas (zoning/ urban planning, highway design, airport management, industrial/ environmental noise regulations, technological improvements in vehicle engine/ tire noise). Future changes in of this nature will be highly influenced by the political process.
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	Low Effects on the health of the individual are typically chronic in nature and noise-induced effects are not recognized as contributing to death or other illness, even if a chronic condition is exacerbated (e.g., a cardiovascular condition).
extent to which risks are currently reduced through in-place regulations and controls	Significant Workplace noise regulations Noise ratings on consumer items / appliances Noise controls on vehicles Vehicle inspection / maintenance Airport planning Noise / nuisance ordinances Road design, materials, location Noise barriers Etc.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
large business/industry	Moderate, occasionally significant in localized areas

small business industry	Moderate, occasionally significant in localized areas
Transportation	Occasionally significant to high: Commercial airports Freight / commuter rail Highways / interstates, particularly at toll plazas, hills / grades and traffic lights Truck terminals and large / small industry serviced by large and / or over-the-road trucks
Residential	Generally low, but with the potential for occasional annoying exceptions (speeding cars, noisy neighbors, leaf blowers, lawn mowers)
Agriculture	Very low
Recreation	Occasionally very high: Sporting venues / automobile race tracks Theater / entertainment districts Associated vehicle traffic for spectators / patrons coming and going
Resource extraction	Intermittently significant near quarrying operations
Government	Nil, moderate near military installations, intermittently significant near air bases
natural sources	Nil
Contaminated sites	Nil
diffuse and non-NJ sources	
Sediment	Not applicable

Soil	Not applicable
non-local air sources (including deposition)	Not applicable
biota sinks	Not applicable

Health effect{tc \l 2 "Health effect"}	Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population affected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
Noise induced hearing loss	*Not included in assessment*			
Sleep disturbance	3	3	5	
Direct impact on other (non-aural) physiological systems such as the cardiovascular system	2	2	5	2
Psychological impact from sleep deprivation or other subjective responses to noise	3	2	5	2
Interference with communications, particularly telephone conversations	1	2	3	1
				2

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Ozone (Ground Level)**
Description of stressor (including etiology)	Ozone is only one of a class of compounds, known collectively as photochemical oxidants, that result from such reactions. The mixture of pollutants that forms on high ozone days is commonly referred to as smog. Ozone is not typically emitted directly into the atmosphere but results from a series of reactions between nitrogen oxides and volatile organic compounds in the presence of sunlight. As a result, exposure to high concentrations of ozone is a problem on hot sunny summer days and the vast majority of exceedances of the National Ambient Air Quality Standards (NAAQS) occur between May 15 and August 31. While elevated concentrations of ozone in the lower atmosphere are considered a problem, the opposite is true for ozone in the upper atmosphere, or stratosphere. Stratospheric ozone, which helps to shield the earth's surface from high levels of ultraviolet radiation, will not be considered here.
Stressor-specific impacts considered including key impacts	Ozone directly affects the respiratory system, where it reacts with and irritates the mucous membranes of the nose, throat and airways; 90% of the ozone inhaled into the lungs is never exhaled. The symptoms which occur at the lowest adverse levels of ozone exposure relate to restriction of airways and include reductions in respiratory volume and velocity (Hortsman, et al. 1990; McDonnell, et al. 1991; Spektor & Lippmann 1991) which can compromise respiratory function in those with existing respiratory diseases as well as facilitating asthmatic reactions in sensitive individuals. Other symptoms at somewhat higher levels of exposure include coughing, chest pain, and throat irritation (Follinsbee, et al. 1988). Exposure to ozone can also increase susceptibility to respiratory infections (Devlin, et al.1991). Repeated exposures to high ozone levels may lead to permanent scarring of lung tissue, loss of lung function, and reduced elasticity (Chang, et al.1992).
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	1. Inhalation of ozone in Outdoor Air
Population(s)/ecosystem(s) exposed statewide	All residents of the state are potentially exposed during the summer months. Tourists, especially at the shore, are also exposed.
Quantification of exposure levels statewide (include indoor air as separate category as appropriate)	In 1999, there were 10 days on which the peak 1-hour ozone concentration exceeded 120 ppb at one or more locations in the state. There were also 46 days on which the peak 8-hour concentration exceeded 80 ppb at one or more

	locations. The maximum 1-hour concentration was 157 ppb. On an average day, the peak 1-hour ozone concentration in New Jersey was in the range of 50 to 65 ppb.
Specific population(s) at increased risk	1) Children are most at risk from exposure to ozone because they are active outside, playing and exercising, during the summertime when ozone levels are at their highest. See Spektor & Lippmann (1991) and Berry, et al. (1991) for studies done at camps in NJ. 2) Adults and children with respiratory illnesses, such as asthma, bronchitis and emphysema, can experience a reduction in lung function and increased respiratory symptoms, such as chest pain and cough, when exposed to relatively low ozone levels during periods of moderate exertion. 3) Adults who are outdoors and moderately active during the summer months, such as outdoor workers and people engaged in sports or other exercise, are also among those most at risk.
Quantification of exposure levels to population(s) at increased risk (due to factors other than exposure) (include indoor air as separate category as appropriate)	Sensitive individuals are just as likely as the rest of the population to be exposed to the ozone concentrations described above under quantification of exposure levels statewide.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	The USEPA has set public health-based National Ambient Air Quality Standards (NAAQS) for ozone at 120 ppb for a 1-hour period and 80 ppb for an 8-hour period. The basis of these standards is thoroughly described in the Ozone Criteria Document (USEPA,1996). The USEPA projects that achieving the 8-hour standard would lower, put not eliminate, the incidence of: a) lung function decrement in children; b) moderate to severe coughs and chest pain in children; c) incidence of lung inflammation in children; and d) emergency room visits and hospitalization for asthmatics.
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk, upper percentile population risk, etc.)	1) Regarding the 1-hour standard, the number of days exceeding the standard was as high as 45 in 1988 and down to only 10 days in 1999. Using this information, we have estimated the NJ population potentially exposed to ozone above the 1-hour standard on at least one day in each of the following years.[1] 1999: 7.6 million 1998: 1.2 million 1997: 3.9 million 1996: 0.6 million 2) The entire population of the state has been potentially exposed to ozone concentrations above the 8-hour standard

[1] **To calculate this exposure parameter, NJDEP ozone air monitors were each assumed to represent one or more counties (a reasonable assumption given the regional behavior of this pollutant) and the population of each county was estimated by extrapolating from 1990 census data to the year of interest (with the slope of the line being the change in population between 1980 and 1990).**

	for decades. In 1999, the 14 ozone monitoring sites recorded 6 to 25 days each above the 8-hour standard.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	A significant proportion of hospital and emergency room admissions can be associated with elevated exposure to ozone. For example, 13-15% of the variability of asthma visits in NJ was attributed to high ozone by Cody, et al. (1992). Weisel et al. (2000) reported that 4-7% of hospital and emergency room admissions for asthma in NJ were associated with ozone levels during June-August 1995.
Size of population(s) affected	a) The entire population of NJ is potentially affected, with those who are active outdoors being more susceptible. b) In 1996, there were about 440,000 asthmatics in NJ and 430,000 persons with chronic bronchitis (American Lung Association, 2000). c) In 1996, asthma was responsible for 16,265 hospitalizations in NJ (DHSS, 2000).
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	M. There have been thousands of studies done on ozone exposure (over 3000 peer reviewed studies were reviewed for the Ozone Criteria Doument (USEPA, 1996)), making this one of the better understood pollutants. However, the weight of the health effects evidence indicates that there is no threshold concentration for the onset of biological responses (CASAC, 1995).
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	M. The current work relating the experience of asthmatics when exposed to high concentrations of ozone may continue to push the boundary below which adverse effects have been experienced.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	+ Precursor emissions should decrease as a result of the actions contained in our current State Implementation Plan for meeting the Ozone NAAQS, but this will not be enough to meet either the 1-hour or the 8-hour standard.
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L. Since ozone is not emitted directly, a catastrophic event is highly unlikely.
Extent to which risks are currently reduced through in-place regulations and controls	In the 1980s, the number of days that the 1-hour ozone health standard was exceeded in NJ ranged from 18 to 60 per year, with most years having above 30. In the 1990s, the number of exceedance days has dropped to 6 to 26 with most years having less than 20. The overall decrease in the number of exceedance days can be attributed to reduction in emissions from automobiles and industrial sources, and control of emissions of gasoline during refueling - all programs implemented by the NJDEP. (NJDEP, 1998)
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	*The following allocations are based on the Inventory of VOC and NOx Emissions in 1996 which has been prepared in support of the State Implementation Plan. (NJDEP,2001).*
Large business/industry	M-VOC / M-NOx

Small business industry	H-VOC / L-NOx
Transportation	H-VOC / H-NOx
Residential	H-VOC / L-NOx
Agriculture	L-VOC / L-NOx
Recreation	M-VOC / M-NOx
Resource extraction	N/A
Government	M-VOC / L-NOx
Natural sources	L-VOC / L-NOx
Contaminated sites	L-VOC / L-NOx
Diffuse and non-NJ sources	
Sediment	N/A
Soil	N/A
Non-local air sources (including deposition)	M: Reduction of both in-state and out-of-state emissions of ozone precursors will be necessary to achieve the Ozone NAAQS in NJ.
Biota sinks	N/A

Human Health Issue Summary: Ozone (ground level)

What is it?
Ozone is one of a class of compounds called photochemical oxidants that result from chemical reactions between various nitrogen oxides (NOx) and volatile organic compounds (VOCs) in the presence of sunlight. Motor vehicle exhaust is a primary source of NOx and VOCs. Inhalation of ground-level ozone has been associated with a variety of respiratory problems, especially asthma, but also including acute and chronic bronchitis, chronic obstructive pulmonary disease (COPD), reduced lung function, and premature death.

What's at risk?
All residents statewide are potentially exposed during the summer months. Children may be at increased risk of exposure because they are active outside during the summer, when ozone levels are at their highest. Adults and children with respiratory illnesses, such as asthma, bronchitis and emphysema, and adults who are active outdoors during the summer are also at higher risk.

Issue: Ozone (Ground Level)
Author: Joann Held
Version: 03/07/01

What are the human health impacts in New Jersey?
Thousands of studies on ozone exposure indicate that there is no minimum threshold for triggering respiratory responses and a significant proportion of hospital visits can be associated with exposure to elevated ozone levels. Federal health-based standards for ozone are set at 80 ppb measured over an 8-hour period, and 120 ppb for a 1-hour period. In 1999, one or more locations in New Jersey were in violation of the 8-hour standard on 46 days, and the 1-hour standard on 10 days. On an average day in 1999, peak 1-hour concentrations were in the range of 50-65 ppb. In New Jersey, there are more than 440,000 asthmatics and 430,000 persons with chronic bronchitis, who may be adversely affected by ozone levels.

What's being done?
During the 1980s, the 1-hour ozone standard was exceeded in New Jersey more than 30 times per year. In recent years, the standard is exceeded much less often—less than 20 times per year. This overall reduction in ozone levels can be attributed to reductions in allowable emissions from automobiles and industrial sources, and by controlling releases at fuel pumps.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
M/4	H/5	Y/5	H/4

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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Particulate Matter (PM)**
Description of stressor (including etiology)	Particulate matter (PM) is a mixture of solid matter and liquid droplets from anthropogenic or natural processes. Composition, size and concentration vary according to sources, geography, seasonality and time of day. Particulate matter is composed of a broad class of chemically diverse particles that range in size from 0.005 microns in aerodynamic diameter (µm) to 100 µm. (NJDEP, 1998). Particles can be divided into several size categories: Total Suspended Particles (TSP) includes all particles; Inhalable particles (PM_{10}) includes all particles smaller than 10 µm in diameter; and Respirable particles (PM2.5) are all particles smaller than 2.5 µm in diameter. Another useful categorization is that of coarse, fine and ultra-fine particles. While the precise size cut points and required percentage of collected particles at a particular size may vary, in general, it could be said that coarse particles are greater than 1 µm in diameter; fine particles refer to PM that is less than 1 µm in diameter; and ultra-fine is used to describe particles that are less than 0.1 µm in diameter. The Environmental Protection Agency (USEPA) uses the term coarse for any particles greater than 2.5 µm and fine for particles less than 2.5µm (http://www.epa.gov/ttn/oarpg/naaqsfin/pmhealth.html 7/2002). Mechanical processes, as well as weathering and combustion, generate the majority of coarse particles. Coarse particles have a short residence time in the atmosphere. Fine and ultra-fine particles are often referred to as secondary particles because many of them are formed from atmospheric chemical reactions. These smaller particles have long residence times in the atmosphere and are able to be transported long distances. Coarse particles are responsible for most of the mass of TSP, but the smaller particles are a larger component of overall particles when particles are quantified based on number.

Description of stressor (including etiology)	Background geogenic and biogenic emission sources of course particles include: wind blown dust from erosion and re-entrainment; sea salt; forest fires; and biologicals such as pollen and pollen fragments, animal skin, scales, secretions, fungal spores, hyphae, and bacteria (USEPA, 1996; HEI, 2002). These biologicals can be a significant portion of PM in certain indoor environments. Naturally occurring fine particles can be generated by the long range transport of dust from the Sahara Desert, particles formed from the oxidation of sulfur compounds emitted from oceans and wetlands, the oxidation of NO_x from natural forest fires and lightning, and the oxidation of hydrocarbons emitted by vegetation. Some common anthropogenic sources of coarse particles include agricultural and mining activities, fugitive dusts from roads, industry, agriculture, construction, demolition, and fly ash from combustion. It is estimated that fugitive dust constitutes about 90% of the estimated PM_{10} emissions in the United States (USEPA, 1996). Only a small percentage is emitted in the fine particle mode. Major anthropogenic sources of fine particles include fossil fuel combustion by electric utilities, industry and motor vehicles; incineration; vegetation burning; and the smelting or other processing of metals. Fine particles can also be formed from the condensation of volatiles that arise from combustion sources. Generally, coarse particle composition is made up of insoluble minerals from the earth's crust (alumino-silicates, iron oxides, calcium oxides, etc.) sea salts, and biologicals. Fine and ultra-fine particles usually consist of a carbon core containing a diverse and complex mixture of metals, ions (sulfates, nitrates and acidity), reactive molecules (ozone, peroxides, aldehydes), hydrocarbons and secondary particles. Secondary particles make up a large component of the ion fraction and include nitrogen oxides (NO_x), sulfur oxides (SO_x), free radicals, other reactive molecules and secondary organic compounds (volatile and semi-volatiles can adsorb onto particles) (HEI 4/02). Fine particles can also adsorb other toxicants from ambient air. The major precursors of secondary particles in the Eastern US include sulfur dioxide (SO_2), nitrogen oxides (NOx), and certain volatile organic compounds. The formation of secondary PM is seen as smog or haze in the eastern U.S. Since smog formation increases with sunlight and temperature, secondary PM peaks during the summer months in New Jersey.
Stressor-specific impacts considered including key impacts	Particle size, concentration and composition all impact the ability of PM to cause adverse health effects. Most coarse size particles are cleared from the nose and upper airways through mucus flow, sneezing and coughing. If the mucus is swallowed, then the particles can be transported into the gastro-intestinal system where they potentially can become absorbed. Fine and ultra-fine particles can be inhaled deep into the lungs, where they can deliver toxic constituents to the lung tissue and blood stream in addition to being lodged in lung tissue and interfering with lung function. If a particle reaches the air exchange region of the lung, the alveolar region, it may never be cleared from the body. For this reason, recent sampling emphasis has been placed on $PM_{2.5}$ (NJDEP, 1998). The observed human health effects of PM include aggravation of existing respiratory and cardiovascular diseases, alterations in the body's defense system against inhaled materials and organisms, and damage to lung tissue. Epidemiological studies show consistent positive associations between exposure to ambient PM and health effects, including mortality and morbidity. (Pope et al., 2002; Samet, et al., 2000; Hong, et al., 2002; Moolgavkar, 2000; Nevalainen, and Pekkanen, 1998). The observed associations of ambient PM exposure with health effects are usually adjusted for the effects of other environmental and socio-demographic factors, and for whether the effects are acute or chronic, in order to quantitatively assess the risk that can be attributed to PM exposure.

	Estimates of PM health effects have shown reasonable quantitative consistency in different studies, with only modest sensitivity to different methods of analysis. It appears that particulate exposure causes an inflammatory response which may damage the body's defense mechanisms (HEI, 2002). Cardiovascular effects from particulate exposure include an increase in coagulation of the blood due to the release of certain molecules in the body. There is also some evidence that lead researchers to believe that particles can move from the lung tissue to other target sites which cause effects at other locations. Deposition of particles leads to stimulation of the nervous system, which can cause a change in breathing and heart rate. While the epidemiological data provide support for the associations mentioned above, a definitive understanding of underlying biological mechanisms has not yet emerged. (USEPA, 1996).
Exposure routes and pathways considered (include indoor air as appropriate)	Inhalation is the exposure route for both outdoor and indoor environments. While outdoor PM concentrations are measured routinely, indoor PM concentrations and composition are not as well known and may be significant contributors to adverse health effects. Both fine and coarse particles can enter homes and other microenvironments, with fine particles penetrating indoor environments more easily than coarse particles. Particulate matter concentration can decrease through gravitational settling and electrostatic attraction. The larger particles settle more rapidly than the smaller ones. (USEPA, 1996). In indoor microenvironments, PM penetrates from the outdoors, and is also generated from indoor sources (cooking, smoking, vacuuming, dusting, walking, etc.) causing indoor concentrations to be greater than outdoor concentrations. Therefore, indoor sources need to be considered to accurately assess total exposure (USEPA, 1996).
Population(s)/ecosystem(s) exposed statewide	All
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	Inhalable Particles Since 1990, New Jersey annual averages for inhalable particulate matter (PM_{10}) have not exceeded the National Ambient Air Quality Standard (NAAQS). Fifty (50) $\mu g/m^3$ is the primary (health) and secondary (welfare) standard. One site in Elizabeth exceeded the twenty-four hour NAAQS in 2000, (156 $\mu g/m^3$ vs. the standard of 150 $\mu g/m^3$), with the second highest concentration in Elizabeth at 108 $\mu g/m^3$. Presently the state monitors for PM_{10} with ten samplers at eight locations around the state (between two sites in Camden there are three samplers).

Specific population(s) at increased risk	Respirable (fine) Particles ($PM_{2.5}$) In the year 2000, there were eighteen locations with twenty-one samplers around the state. The annual arithmetic mean concentration exceeded the annual NAAQS of 15 $\mu g/m^3$ in eight locations. However, only two of those locations had enough data to calculate a valid annual arithmetic mean. The twenty-four hour average NAAQS of 65 $\mu g/m^3$ was exceeded in four locations in the year 2000. In addition, $PM_{2.5}$ is measured by an alternate monitoring method at four locations around the state. This method measures $PM_{2.5}$ continuously. The annual NAAQS was exceeded at one location for 2000. The 24-hour average NAAQS was not exceeded in any of the four locations. Non-ambient conditions, mainly indoors at home or at work, occupy the vast majority of a person's time. In the U.S., the average daily time spent indoors is 20 h/day, or ~83% of the day. Some additional time, about 1.0 to 2.0 h (~6%) of the day, is also spent in other non-ambient microenvironments (e.g., in vehicles in transit) (USEPA, 1996). Therefore, ambient monitoring of PM may not be representative of the total exposure for people who work/reside in New Jersey. Asthmatics show increased response to PM, specifically acid aerosols and bioaerosols (USEPA, 1996). Asthmatics have been shown to be more responsive than non-asthmatics to acid aerosols in controlled exposure studies. It is generally accepted that Asthma exacerbations are associated with outdoor and indoor bioaerosols. Chronic Obstructive Pulmonary Disease (COPD) patients show increased PM deposition and impaired clearance. Airway inflammation or compromised immune status may alter the tissue's ability to respond to inhaled particles. Susceptible groups most clearly at special risk for PM effects include the elderly, the young, and those with cardiopulmonary disease. Particulate matter may impair breathing among asthmatic and COPD patients. Epidemiological findings indicate that ambient PM exposures are also associated with increased risk for mortality and hospitalization due to cardiovascular causes. Cardiac arrhythmia has been hypothesized as being involved in mortality due to acute PM exposure. Epidemiological studies also indicate that the risk of mortality and morbidity due to lower respiratory disease (e.g., pneumonia) increases with increases in levels of ambient PM. This may be due to exacerbation of pre-existing respiratory disease. PM may also increase susceptibility to infectious disease. The epidemiological findings also indicate that individuals with preexisting infectious respiratory disease (e.g., pneumonia) are at increased risk for PM effects. Smokers constitute a significant fraction (ca. 80%) of individuals with COPD and a smaller but notable portion of cardiovascular disease patients. Therefore, smokers are another subpopulation that is at increased risk for PM health effects. Children and adolescents may also be potentially susceptible to PM effects due to their increased ventilatory frequency resulting in greater respiratory tract PM deposition. In children, epidemiological studies reveal associations of PM exposure with increased bronchitis symptoms and small decreases in lung function. (USEPA, 1996). The Health Effects Institute reported on a recent study by Goldberg which found that diabetics are sensitive to PM exposures (HEI, 4/2002). They also cited other studies which suggested that pregnant women, fetuses and newborns are more susceptible than the general population to PM derived health effects (Woodruff, et al., 1997, HEI, 4/2002).

Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	Exposure (particularly to fine particles, which are subject to long-range transport,) is similar for most residents of the state. However, there do seem to be elevated levels of $PM_{2.5}$ in urban environments (Chuersuwan, 2000) which is probably related to local sources.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	Researchers from the Johns Hopkins Bloomberg School of Public Heath have analyzed air pollution, weather and mortality in 90 large cities across the United States as part of the National Morbidity, Mortality and Air Pollution Study (NMMAPS). The estimated increase in the relative rate of death from cardiovascular and respiratory causes is approximately 0.2% for each increase in the PM_{10} level of 10 µg per cubic meter (Samet et al., 2000 and www.jhsph.edu/Press_Room/air%20pollution%20update.html). Other researchers have assessed long-term exposure to fine particulate and they found a relative risk increase of 4%, 6% and 8% for all-cause, cardiopulmonary and lung cancer mortality, respectively, for each 10 $\mu g/m^3$ increase in fine particulate (Pope et al., 2002).
Risk Characterization Risk estimate(s) by population at risk including probability and number of	The National Ambient Air Quality Standards were promulgated to protect sensitive populations. From this point of view, it stands to reason that asthmatics, elderly, smokers, and children would be at increased risk from exceedances of the standard. New Jersey currently meets the PM_{10} concentration standards. Specific health impacts from potential violations of the new health-based $PM_{2.5}$ standard are uncertain and it is difficult to quantify them at this time. Nonetheless, some studies suggest that any increase in ambient $PM_{2.5}$ concentration (even below existing standards) leads to increases in both symptoms and mortality. While specific numbers are difficult to come by, estimates of increased respiratory and cardiovascular symptoms and mortality associated with particulate matter from all sources have been made with a variety of confidence levels. It is unclear whether the new standard for $PM_{2.5}$ is truly protective for sensitive subpopulations. Further research is needed to adequately assess the new standard.

cases/occurrences (Specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	Abt Associates, Inc. was commissioned by the USEPA, Office of Air Quality, Planning and Standards, to reanalyze data and develop equations which estimate premature mortality due to outdoor levels $PM_{2.5}$ (USEPA, 1999). Separate equations were developed from two major studies, Pope et al., 1995, and Dockery et al., 1993, because each study has it's own strengths and weaknesses. There are many uncertainties associated with these equations and their application to New Jersey levels of $PM_{2.5}$ and population. The estimates stated below use the difference between the average of the monitored values for the state and the NAAQS standard in the equations. This assumes that the NAAQS is protective of human health. This introduces uncertainty because researchers surmise that no level of $PM_{2.5}$ exposure is safe. Other assumptions and limitations which increase uncertainty include but are not limited to assuming that: A certain percentage of the population of NJ matches the age group and demographics of the original studies; Ambient mean and median $PM_{2.5}$ levels from the original studies represent $PM_{2.5}$ exposure; Confounders such as other air pollutants, nutrition, lifestyle, health status and other stressors are insignificant; Conditions in the geographical location and years of the original studies represent current conditions in NJ; The statistical programs used to generate the results functioned properly, The appropriate models were applied to the data; Relative risk can be extrapolated to absolute or measured risk; and Abt Associates correctly derived the equations. Application of the equations derived by Abt Associates, Inc., results in an estimate of approximately 500 to 1000 premature deaths per year in NJ due to $PM_{2.5}$. These estimates need to be interpreted with their associated limitations, assumptions and uncertainties.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	For most individuals, effects are small and difficult to attribute to specific environmental conditions. Effects are generally exacerbations of existing conditions. Severity of effects varies from mild to severe asthma episodes to increased severity of pulmonary infection, depending on specific conditions.
Size of population(s) affected	Based on CDC estimates, there are 540,000 asthmatics in NJ. NJDHSS statistics for 1997 show 2,775 deaths from chronic obstructive pulmonary disease in NJ.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	M - The ambient air sampling data for PM_{10} is a long term, high quality database but since sampling is conducted on a pre-determined schedule, some high concentrations may be missed. Ambient air sampling for $PM_{2.5}$ has been in place in New Jersey since 1999. Both Samet et al., and Pope et al., used epidemiology and found associations between increases in mortality and increases in PM concentrations. Some of the major uncertainties in these large epidemiological studies include biased air pollution effects due to confounders, the shape of the dose-response curve, and the statistical methods and software used for such data intensive analyses.

Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	M – Ongoing monitoring of fine particles will increase the potential to determine a significant change in the risk estimate from exposure to PM.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, -,--,--- where + is improvement)	. 0. It is difficult to be certain if the risk will decrease or increase in the short term. The EPA is moving towards relaxing the New Source Performance Standards (NSPS). This standard requires facilities to upgrade their pollution control technologies when any changes are made at that facility. This has an enormous potential to impact the air quality in New Jersey since much of our PM is generated from power plants located in other parts of the country. If the NSPS are upheld, and the utility plants upgrade their systems, the air in NJ could see a significant reduction in PM. However, since the EPA is not supporting the NSPS, it seems that there will not be any improvement in ambient levels of PM from the utility sector for the state of NJ. Additionally, motor vehicle miles driven are increasing and mobile sources are a significant source of PM.
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	M/L Catastrophic events significantly impacting ambient PM concentrations are of a medium to low likelihood. However, since NJ is in a drought, forest fires could significantly increase PM in the state if the event were to happen upwind or in close proximity. Also, any terrorist attacks which cause massive combustion or release of bioaerosols, could significantly impact the concentrations of PM if the event were to happen upwind or in close proximity.
Extent to which risks are currently reduced through in-place regulations and controls	NAAQS standards for particulate matter regulate levels of ambient particulate matter. Industrial facilities, as well as the automotive sector, have limits on the amounts of PM released. Large industrial facilities currently remove the majority of particulates from the exhaust gas prior to the gas being released to air. Much of the potential pollution is currently captured before release. Such was not the case before the Clean Air Act. Severe pollution episodes seen in earlier years are not observed now, and even those episodes which we consider severe using today's standards, are relatively mild compared with those experienced before today's emission standards. Towards the end of 2000, EPA proposed new standards for diesel and gasoline fuels. These should reduce particulate matter even further. While there are indoor PM standards for the work place, there are no regulations that control particulate matter in indoor residential environments where people spend most of their time. Regulations providing smoke-free environments have helped reduce PM in commercial facilities.

Relative Contributions of Sources to Risk (H,M,L)	The chemical complexity of airborne particles requires that the composition of primary and secondary components be considered when attempting to identify sources. Uncertainties in emissions inventory estimates could range from about 10% for well-defined sources (e.g., for SO_2) to an order of magnitude for widespread and sporadic sources (e.g., fugitive dust). Receptor modeling has proven to be a useful method for identifying contributions of different types of sources, especially for the primary components of ambient PM. Apportionment of secondary PM is more difficult because it requires consideration of atmospheric reaction processes and rates. Results from eastern U.S. sites indicate that stationary combustion and fugitive dust are major contributors to ambient PM samples in the East. Sulfate and organic carbon are the major secondary components in the East. Therefore different control strategies are likely to be needed, depending on whether fine or coarse particles (or both) are selected for control.
Allocation of stressor-specific risk to primary NJ sources	For those categories for which the assessment isn't necessarily obvious, there should be at least a sentence explaining the reason for the H,M,L assignment
Large business/industry	High
Small business industry	Medium
Transportation	High
Residential	Medium/High
Agriculture	Medium/Low
Recreation	Low
Resource extraction	Low
Government	Low

Natural sources	Low
Contaminated sites	Low
Diffuse and non-NJ sources	
Sediment	Low
Soil	Low (soil-derived dust is not a big factor in the urban environment except in the vicinity of construction sites.)
Non-local air sources (including deposition)	High for fine particles and low for coarse particles Dry deposition of fine particles is slow. Nuclei-mode (ultra-fine) particles are rapidly removed by coagulation into accumulation-mode particles. Accumulation-mode particles are removed from the atmosphere primarily by forming cloud droplets and falling out in raindrops. Coarse particles are removed mainly by gravitational settling and inertial impaction. Primary and secondary fine particles have long lifetimes in the atmosphere (days to weeks) and travel long distances (hundreds to thousands of kilometers). They tend to be uniformly distributed over urban areas and larger regions, especially in the eastern United States. As a result, they are not easily traced back to their individual sources. The formation of sulfates from sulfur dioxide emitted by power plant plumes can occur over distances exceeding 300 km and 12 hour transport. Nitric acid also can be formed in these plumes. Similar transport can occur in urban plumes. The residence times of fine particles can be long. Therefore transport distances of several hundred to several thousand km are possible. (USEPA, 1996). The regional homogeneity is an indication that the eastern US $PM_{2.5}$ is composed of secondary aerosol that is produced several days after the emission of its gaseous precursors. The excess $PM_{2.5}$ concentration in urban centers suggests that primary emissions such as auto exhaust and heating furnaces are responsible for much the urban $PM_{2.5}$ hot spots. (USEPA, 1996). Coarse particles normally have shorter lifetimes (minutes to hours) and only travel short distances (<10's of km). Therefore, coarse particles tend to be unevenly distributed across urban areas and tend to have more localized effects than fine particles. (Dust storms occasionally cause long range transport of the smaller coarse-mode particles.) (USEPA, 1996).
Biota sinks	Low

Issue: Particulate Matter
Author: Linda Bonanno
Version: 05/27/02

What is it?
Particulate matter (PM) are solid particles or liquid droplets from smoke, dust, ash or liquid vapor that can remain airborne for long periods of time. Particulate matter results from all types of combustion, materials abrasion, and re-suspension of dust. Bioaerosols, which include plant pollen, animal dander, molds and yeasts, bacteria, and viruses, may be particularly high indoor contributors to PM exposures. Particulates are usually measured in two size ranges. Coarse particles (between 2.5 and 10 microns in diameter) are formed as a result of crushing or grinding (e.g., in mining operations), and include the bioaerosols. Fine particles (less than 2.5 microns) result from condensation of volatile combustion products. Fossil fuel combustion, (vehicles, power utilities, and industry), burning of vegetation, and metal smelting are sources of fine particulates. Inhalation can aggravate existing respiratory and cardiovascular disease, damage lung tissue, and interfere with lung function. Increases in PM exposure are also associated with increased daily mortality, although the exact cause is uncertain.

What's at risk?
Groups most widely affected include young children, asthmatics, the elderly, smokers, and individuals with chronic lung or cardiovascular disease. Asthmatics show increased response to acid aerosols, and bioaerosols. Smokers constitute approximately 80% of individuals with Chronic Obstructive Pulmonary Disease (COPD), and a portion of cardiovascular disease patients. Children and adolescents may be at increased risk because they have higher respiration rates.

What are the human health impacts in New Jersey?
The entire state is currently in compliance with federal standards for coarse particles (PM 10). Recent studies have shown that fine particles (PM 2.5) may be of greater concern. Fine particulates are inhaled deeply into the lungs, where they become lodged and interfere with lung function. In contrast, PM 10 are cleared fairly rapidly from the nose and upper airways by sneezing and coughing. New standards for PM 2.5 are being developed. The average American spends about 20 hours per day indoors. Cooking, smoking, dusting, vacuuming, and walking on carpets are all sources of particulates to which people are exposed daily. For most individuals, the effects are small and difficult to attribute to specific environmental conditions. Typically, the effect is a worsening of an existing health problem.

What's being done?
Recent research has turned toward the significance of the smaller (PM 2.5) particles, and their relation to illness. Controls are in place on large industrial facilities, and new standards for auto fuels have been developed, which are expected to further reduce PM 2.5. There are indoor PM standards for the workplace, but no regulations targeting residential exposures.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population affected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
H	H	Y	H
5	5	5	5
			H 5

Issue: Particulate Matter
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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	

Stressor Description of stressor (including etiology)	**Pesticides-Food** Pesticides are any substance or mixture of substances employed to destroy, prevent or control pests. By design, the very nature of pesticides creates some risk of harm to humans, animals, plants, living organisms, or environment. Approximately 600 registered unique pesticidal substances are unified only by this characteristic. These substances have greatly different chemical, physical, and toxicological (acute and chronic) characteristics, differing environmental fate that determines movement and persistence. About 300 pesticides are potentially used in all aspects of food production. Pesticides may be commonly classified into broad groups according to the pest to be controlled. For example, insecticides control insects, herbicides control plants and weeds, fungicides control molds or fungal growth. There are also pesticides that are insect- or plant- growth regulators, anti-microbials, among many others. Pesticide also can be classified on the basis of general chemical characteristics. Pesticides of a similar class may be associated with certain symptoms or a common mechanism of action. The broad chemical pesticide classes includes halogenated organics (DDT, methoxychlor, methyl bromide), chlorinated cyclodienes (dieldrin), (inorganics (sulfur, arsenicals), metals (copper-containing substances), organophosphates (OPs), pyrethroids, carbamates, phenoxy-herbicides acids and esters, chlorinated fumigants (methyl bromide, dichloropropane), chloroacetanilides, triazines, and substituted ureas. Other classifications may include microbiologically based (Bt endotoxin) or biologically-derived substances (pheromones, growth regulators, botanical oils) among many others. There are over 300 unique pesticides that are labeled for use on designated food crops/commodity. If used properly, each pesticide will have an established tolerance, the legal limit for the maximum allowable pesticide residual on that crop or commodity. A residual will include the pesticide, any toxicologically significant degradation product, metabolite, or any impurity. A certain fraction of these pesticide residuals will enter the food chain and be consumed.
	Historical Use Pesticides are those pesticides that include the banned or canceled long lasting chlorinated cyclodienes and DDT- type pesticides that were widely and heavily used in agriculture and household soil and termiticide treatments. Many of these pesticides are among those known as persistent organic pollutants (POPs). The POPs no longer in use include chlordane, heptachlor, aldrin, endrin, and dieldrin. Dieldrin is very toxic and extremely persistent and all uses cancelled since 1987. Chlordane and heptachlor have had all uses canceled since 1988. Chlordane and it's breakdown product oxychlordane; and other associated components such as octachlor and nonachlor are commonly found. Heptachlor, and its toxicologically active breakdown product heptachlor epoxide are also commonly found. Chlorinated 4,4'-DDT and degradation products DDD and DDE were also heavily used both agriculturally and domestically for insect control with many uses cancelled in 1972. It is

	because of their persistent nature, potential for transport, and being found throughout the world wherever they are monitored, that these POPs are all under current international negotiations.
stressor-specific impacts considered including key impacts	Exposure in humans to the various pesticides cause a vast range of effects. The severity of effects range from sub-threshold to clinically observable, acutely mild to moderate effects to gross and severe impacts that may result in death. The duration of symptoms span the range of time from the briefly acute, to episodic, to chronic and persisting throughout a lifetime. Pesticide symptoms may include headache, dizziness, weakness, nausea or vomiting; dermal /skin or eye irritation or burning sensation; sweating, respiratory distress, slow or irregular heartbeat, twitching or tremors, to serious or long-term endocrinal, neurological, developmental, and reproductive disorders, including fetal and birth defects. Acute delayed and subchronic neurotoxicity and multigenerational effects also may be observed. Long term or chronic exposure to certain pesticides may be associated with tumor or cancer formation. Certain chemically related pesticides may have a common mechanism of toxicity such as organo-phosphates (OPs). The hazards of 40 individual OPs are currently being comprehensively assessed on a case-by-case basis (EPA, NTP). Upon exposure, all OPs are neurotoxic, causing cholinesterase inhibition and related clinical symptoms, up to and including death. (EPA & HIARC 1997,1998). All are considered highly poisonous, except for acephate and malathion which are classified as moderately poisonous. Only a few OPs (e.g., parathion, phosmet) are classified as possible human carcinogens, or as causing reproductive effects (e.g., trichlorfon and guthion). OPs are not considered to accumulate and persist in the body. OPs may cause neuropathology of the visual system or effects on cognitive functions such as learning and memory. To address a national concern, evaluating endocrine disruption effects is one of the newest EPA initiatives to be established under the Endocrine Disruptor and Screening Program (EPA Report to Congress 2000). Most pesticides have not been fully evaluated with respect to endocrine disruption. EPA tests and criteria are still being developed (ES&T, October 2000) and have been criticized as deficient in defining and assessing low dose effects of pesticides on the immune system and neurological structure (NTP Panel 2000). Most pesticide testing, evaluations, and assessments are heavily based on or derived from high dose animal testing data.
Exposure Assessment	
exposure routes and pathways considered (include indoor air as appropriate)	For pesticide crop residues in food, the major pesticide exposure path under consideration in this section is through ingestion of residues. Exposure to pesticides through other dietary sources such as ground and surface water is not considered in this section.

Population(s)/ecosystem(s) exposed statewide	There are no crops grown or food consumed that can be guaranteed completely pesticide residue free, so that the NJ population statewide may be exposed to pesticide residues through their diet. Even "organic" foods that allow naturally-obtained pesticides and do not use synthetic pesticides, may have pesticide residues. An exposure may also exist from no longer used, long-lived "historic" pesticide residues taken up by crops grown on soils with pesticide residues (DDT, chlordane, heptachlor, dieldrin). There are statewide sub-populations of significant size that are exposed and considered to be at higher risk. Subgroups may included elderly (those over 65 years old), infants, and children (12 years and under), females/males of reproductive age (13 years +), and women that are child-bearing or nursing.
Quantification of exposure levels statewide	There is a complex sequence of methods that are employed in evaluating specific pesticide risks. In general, EPA's approach for calculating pesticide exposure is based on the product of two variables: 1) amount of food ingested and 2) amount of pesticide residues remaining on the food. At the national level, data are being collected in an attempt to estimate exposure. The USDA collects data on food consumption, major crops acreage, agricultural pesticide usage. Meat, poultry, some egg products are under supervision of USDA/FSIS. FDA collects intra-state and exported commodity (crop) residue data to enforce the legal tolerances established by EPA. FDA conducts the national Total Diet Study and market basket surveys. EPA uses residue & usage data in pesticide re-registration and dietary risk assessments. Some states participate in USDA/AMS and carry out residue testing program raw and processed foods. NJ does not participate in the USDA studies. NJ does participate in collecting certain food crops for FDA analysis under an EPA cooperative agreement but, there is little data available for NJ crops. These national surveys do not routinely include NJ grown commodity foods, such as foods sold at roadside stands, which is commonly consumed by NJ residents. Many different pesticides are used in growing and producing food crops for human consumption. Food monitoring studies are documenting the consistent presence of many different pesticide residuals (pesticide parent, metabolites, degradation products and other impurities) in foods. There are national estimates available from pesticide residue monitoring in selected foods. For example, pesticides were found in about 40% of grain samples, almost 55% of fruits and 30% of vegetable samples. Databases from FDA, USDA and FOODCONTAM (state-based residue monitoring) have indicated an increase in pesticide residue detections over the past decade. This is due in great measure to improved analytical methods and instruments. However, a small percentage of samples have actual legal violations of tolerance levels, that are the legally accepted levels of pesticide residues, and this percentage has been found to be decreasing over the years. A violation occurs when a residue level is too high by exceeding the tolerance, or because that pesticide doesn't have an established tolerance and no use is allowed on that crop.

	NJDEP Pesticide Control obtains agricultural pesticide use data. The roadside stand or self-harvested food commodity residue data has just been initiated as a pilot program in 2000. There are no current NJ data available that can to be used for quantification of actual exposure to pesticide residues from food grown in NJ. All of the historical use POP pesticides can still be found in the NJ environment - especially soil- in spite of not being used for many years. A search of USDA and FDA databases listing pesticide in food detections indicates that these historic use POP residues are still being detected in virtually all types of food products, even those that are consumed on a daily basis. The most commonly found include DDE and dieldrin. Many of these POP food residues accumulate in humans, thus adding to the body's burden of these pesticides. It is difficult to actually quantify the exposure level to the over 300 different pesticides used on foods. Further, many types of effects have not been systematically assessed even for individually suspected pesticides. The conclusion is that while there is much data available, there are great gaps in what is required before a valid assessment can be performed on the impacts from the presence of the myriad of pesticides.
specific population(s) at increased risk	A National Academy of Sciences report (1993) identified infants and children as being more vulnerable to pesticide exposure. Infants and children have higher susceptibility to pesticide residues due to their stage of immature development and their increase in risk from pesticide exposure. Exposure to even trace levels of POPs at crucial times in fetal or infant development can disrupt or damage human hormone, reproductive, neurological, or immune systems. Other specific subgroups that can be affected include nursing mothers and men or women of childbearing years.
quantification of exposure levels to population(s) at increased risk (due to factors other than exposure)	The NAS report examined food consumption data from a variety of sources. Consumption patterns for infants & children differed from adults in three major areas: - children consume more food calories relative to body weight than adults; - dietary variety increases with age (fewer distinct foods); & on a body weight basis, infants & young children consume more of certain foods than adults do, such as milk, applesauce & orange juice. For evaluating chemicals that may cause endocrine disruption, females and males over 13 years of age have also been targeted. Women bearing children or nursing are also a susceptible subgroup. Such studies have not been done for pesticides, let alone pesticide residues found in food. This type of data is not currently available.
Dose/Impact-Response Assessment	
quantitative dose/impact-assessment employed for each population considered	A pesticide's oncogenic potency is expressed as Q*. The Q* is the slope of the dose response curve from animal tests yielding a positive oncogenic response; high values indicate a strong response. Q* values can range from approximately 1 x 10^{-0} to 1.5 x 10^{-7}. Acute toxicity is based on studies required for pesticide registration with appropriate safety factors incorporated.
Risk Characterization	

risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	The Theoretical Maximum Residue Contribution (TMRC) assumes all residues are present on all crops with a tolerance. The TMRC x Q* = excess tumor incidence/unit dose. This is typically small (approximately 10^{-6}), assuming daily exposure at this level for a 70-year period. The tolerance is set using acute toxicity data submitted for pesticide product (and active ingredient) registration. A common uncertainty for non-carcinogens is ten-fold. A pesticide tolerance sets the maximum permissible residue levels based on a negligible risk standard. For carcinogens, an excess of one in 10-6 range is usually used for regulation. Tolerances are set up on a national basis by EPA and enforced by FDA and decisions are made on a risk-benefit basis, that is the risk of pesticide use is compared to the benefits of each pesticide's use. As a first approximation of the exposure, a specific pesticide residue may be assumed to be present at the 100 % tolerance level. This estimate is used as the TMRC and that is compared to a level that is a toxicologically-derived endpoint, the Reference dose (RfD) or other selected acute or chronic reference point for a particular pesticide such as NOAEL. This is a starting point for the EPA as each pesticide is reassessed for new tolerances or criteria for other matrices under the FQPA. Currently in NJ, there is no coordinated way of tracking incidences of pesticide poisonings. The medical system also does not coordinate tracking emergency room visits related to pesticide poisonings. The New Jersey Poison Information and Education System (NJPIES) serves as the Poison Control Center for NJ. Incoming calls are tracked and NJPIES received more than 4500 pesticide related calls in a year, of which 82% originated from a residence. For the pesticide-related calls, ingestion was the most common route of exposure (61%) followed by inhalation (20%). In the future, these data and additionally needed residential information may provide useful indicators of potential residential pesticide exposure and hazards.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Potential effects of pesticides on people of any age include central nervous system damage, increase in the risk of cancer, and respiratory illness. Infants and children may be especially vulnerable to effects on development, on the immune, reproductive and the visual systems. The effects of many pesticides can only be estimated from animal exposure studies at high doses. For certain pesticides effects are considered to be severe, chronic, persistent, and even irreversible. However, with the exception of acute effects, linking chronic outcomes with specific pesticide intakes and exposures within the diet is nearly impossible at this time.

size of population(s) affected Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	Most of the NJ state population and population of the subgroups are effected. High The uncertainties in the existing data are less a concern then the huge data gaps in the overall data and knowledge on all the pesticides. There is lack of knowledge of the effects of simultaneous exposure to many pesticides. The availability of resources for pesticide management are also a hindrance.
	Regulations required the EPA to establish tolerances that "protected public health." Up until 1996, tolerances, TMRC's and other measures did not take into account the additive nature of the various residues, let alone the possible synergistic effects. Now the residues from all forms of exposure (through food, air, water, or dermal absorption) have to be considered. However, there are limited pesticides under this kind of evaluation and assessment. The current focus is on the highest risk pesticides such as organophosphates There are data gaps in the exposure data available for dietary intake. Of 316 pesticides with food tolerances, only 163 of them are routinely analyzed under FDA's MARMS. Pesticide metabolites and breakdown products, significant or toxic inert ingredients need to be analyzed but we do not have estimates of these. No risk estimates can be done because this basic data are not available. There are large data gaps that exist in impacts of pesticides, both acute and chronic effects. Even though FQPA requires the consideration of pesticides with common mechanism of action or toxicity, this data is not available for many of the pesticides. The potential for endocrine disruption, multigenerational effects, chronic impacts on immune and neurological systems, and cognitive learning is not known for most pesticides. While similar acting pesticides are presumed to create additive effects, data on synergistic effects are not known.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	Moderate The passage of the Food Quality Protection Act (FQPA) is bringing a reassessment and new data into the risk-assessing process for pesticides.

potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =, where + is improvement)	+++ There is excellent potential for changing the underlying risk from pesticides in food. The implementation of the FQPA changed the process by providing that tolerances be set to include residues in all types of food (fresh and processed) and that it include all risks, not just cancer. FQPA states that tolerances be "safe" and defined as "a reasonable certainty that no harm will result from aggregate exposure", including all exposure through diet, other non-occupational exposures, and drinking water. The reassessment of crop/food tolerances and implementation of FQPA will afford a better estimate of unreasonable risks and their reduction. Infants and children will be further protected with an additional risk factor; the residue might be lowered for foods consumed heavily by children (e.g., apple juice, milk). Future research and implementation of Best Management Practices (BMPs) bio-intensive IPM programs using biocontrols on major crops would reduce pesticide use and thus reduce risks compared to chemical dependence. Greater control may be obtained by greater use of predators and parasites for pest control. Reduction of human exposure during application and drift could also be realized.
potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	High The main exposure risk that is indicated involves chronic exposure to relatively low amounts of pesticide residues. The potential impact from catastrophic event such as food poisoning caused by pesticides incurring illness or even fatality would be very high. Due to food production and marketing methods in the USA, a catastrophic pesticide problem would have a huge and widespread impact. Fortunately, the likelihood of such an event is extremely low, because of the regulations and restrictions for pesticide use associated with food.
	In the USA, nearly all cases of mortality associated with pesticides have been unrelated to each other, occurring independently in space and time. Chronic poisoning has not been a major problem. This is partly due to the limitation and regulation of pesticides such as mercury, strychnine, arsenic, fluoride, cyanide, and HCB. Hayes & Laws provide a table of acute pesticide poisoning outbreaks documented between 1933 and 1987. Of 115 reports, 28 involved food poisoning, 11 of these included fatalities.
extent to which risks are currently reduced through in-place regulations and controls	The exposure risk presented by pesticides involves chronic exposure to relatively low amounts of pesticide residues through various pathways. Pesticide risks that are national in scope are being slowly addressed under the FQPA schedule. Existing pesticide in food tolerances will be assessed by 2006. Over 9000 commodity / pesticide active ingredient combinations currently have tolerances and will need assessment. Under FQPA, major changes based on national risk assessments were instituted. Aggregate assessment of all non-occupational sources of exposure for each pesticide has to be performed. Furthermore, risks from pesticides with common mechanisms of toxicity have to be aggregated. The current risk assessments being carried out for the high use and high risk organophosphates (OP), such as diazinon, azinphos-methyl (guthion), and methyl-parathion, are resulting in the recommendation and initiation of severe curtailment of use order to lower the newly assessed exposure risks. Chlorpyrifos, one of the highest used OPs, is also being reassessed and homeowner use will be reduced. Additional consideration has to be taken for children's special sensitivity and exposure to pesticides. Under endocrine disruption screening, risks that were exceeded by e.g., the fungicide vinclozolin, are being decreased to reasonable levels by curtailing its use on major crops.

Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
large business/industry	
small business industry	
transportation	
residential	Variable/Low -home garden food production
agriculture	High – food commodity production
recreation	
resource extraction	
government	
natural sources	
contaminated sites	Moderate - site specific; long term risk due to use of toxic and persistent pesticides in NJ.
diffuse and non-NJ sources	
sediment	Variable or unknown risk for consumed fish.
soil	Moderate - site specific; long term risk due to use of toxic and persistent pesticides in NJ.
non-local air sources (including deposition)	Variable/Low effect on exposure from food.
biota sinks	High/Variable risk from pesticide accumulation in fish caught for food consumption.

Human Health Issue Summary: Pesticides - food

What is it? Pesticides are used in agriculture to increase yield and decrease costs. Unfortunately, pesticide residues often make it into the diet of New Jersey citizens. As with other

pesticide health effects, the wide range of pesticides in use makes it difficult to pinpoint individual health endpoints. However, chronic pesticide health effects include nervous system damage, immune, visual, respiratory and cancer effects.

Who's at risk? All New Jersey citizens are exposed to pesticides in the food that they eat. Monitoring of food products shows that 40% of grain samples, 55% of fruits and 30% of vegetables test positive for at least some level of pesticide residues. In a few cases, these detections show concentrations of pesticides over established safety limits. There is evidence that children are more susceptible to pesticide toxicity because of rapid growth during development and the higher body burden that results when children intake levels are similar to adults.

What is the extent of human health problem in New Jersey? Linking chronic outcomes with specific pesticide intakes is nearly impossible at this time. The possibility exists that a significant contamination event could have a huge and widespread impact.

What's being done? Pesticide application is regulated through a federal licensing process. Food is monitored to identify samples of elevated contamination.

Risk Summary
A large population is exposed to possible pesticide contamination, but the levels of exposures are generally low and within established safety limits. These factors led the Technical Working Group to assign a Medium ranking of health risks.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discreter communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
M (2)	M (3)	N*	M (3)
			3

* Some potential for home gardeners to be more exposed to food-borne pesticides.

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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Pesticides-Indoor**
Description of stressor (including etiology)	According to data referenced in USEPA "A Guide to Indoor Air Quality," 80% to 90% of the exposure to pesticides occurs through the indoor environment. Dozens of pesticides have been measured in various studies of residential indoor air. Pesticide exposure in the indoor environment can come from direct application of pesticides by the homeowner. Commonly, insecticides are used for this purpose. Pesticidal products are used as disinfectants. Another important source is professional structural pest control using termiticides to control termites. Persistent termiticides are considered a significant reservoir source of pesticides in air, and may be transported long distances (Jantunen, et al., 2000). Many of the extremely persistent pesticides have not been in use for over a decade, yet they still may be detected in indoor air. Worse, they may be in indoor air at higher levels than those found for outside air. Many studies show widespread presence of pesticide residues in homes. Most levels found are considered a low but significant source of certain pesticides. This is true especially for multiple-use pesticides used in residences and workplaces, in the outdoor environments such as lawn and garden, and on food crops.
Stressor-specific impacts considered including key impacts	The impacts are dependent on the specific pesticide(s) involved. Symptoms may be caused by exposure to the individual pesticide(s), or to the formulated product that includes proprietary emulsifiers, stabilizers, adjuvants, surfactants, solvents, impurities, or other additives. Indoor air pollutants may be associated with a noxious or aversive property and may cause taste and odor problems. Over-exposure to indoor air pesticides may result in dermal or respiratory problems, nervous system dysfunction, headache, fatigue, mental confusion, impaired memory, reduced attention or performance (e.g., decrease in locomotor skills). CNS depression, vertigo, and tremors also are common symptoms of exposure solvents. Commonly reported symptoms include dermal / skin irritation, irritation of mucosal membranes, eyes, nose, throat; and lower airways. Damage to the CNS, the internal organs such as kidneys or liver, or non-specific hypersensitivity reactions are less common.

Exposure Assessment	
Exposure routes and pathways considered **(include indoor air as appropriate)**	The exposure routes of main concern include direct exposure to pesticide residues during indoor or outdoor residential or workplace pesticide applications. Many households in NJ presently use and/or have used residential pest control services employing conventional chemical treatment. Significant exposures result from emission and re-emission pesticides initially adsorbed to surfaces, walls, and especially foam cushions and rugs. Soil or dust track-in from the outdoors or site of application. Rugs and household dust on rugs or floors are a major source of pesticide residues. These sources also serve as a large sink for exposure to infants and children. The route of exposure includes inhalation of pesticide residues in the vapor or aerosol state, dermal (skin) contact and absorption, and exposure from residues carried by soil particulates and dusts. Significant exposures to petroleum hydrocarbon solvents can occur with pesticide application. Solvents are used to dissolve or "carry" the pesticide in the formulated material to be applied and are a source of volatile organic compounds (VOCs). Bukowski, Meyer, et al, (1995, 1996), in studies carried out by the NJDEP, found that levels of VOCs after indoor broadcast treatments approached or exceeded levels at which low level effects would be predicted. Such effects include headaches, dizziness, nausea, etc. In a 1990 survey of households with children under five years of age, about 50% had at least one pesticide product within reach of children. Pesticide exposure by ingestion of dust by small children (under 6 years old) may be of greater importance than the respiratory exposure to pesticides (Lewis, et al.). Household dust has been found in a number of studies to have higher pesticide levels than the surrounding outside soil. It is expected to find residuals around the home from the persistent pesticides that were used. However, even pesticides that easily degrade as evidenced by their short half-lives in soil, degrade much more slowly and increase their persistence once in the indoor environment. The NOPES Program (1986-1988) was one of the first large-scale studies to systematically examine non-occupational household pesticide exposures (32 pesticides). The types of pesticides were found to vary according to locales and pesticide use. For example, the four most commonly found pesticides diazinon, o-phenylphenol, propoxur, and DDVP, were found sporadically only at trace levels in later studies. Thirty commonly used household pesticides were found in house dust and yard soil. Generally, pesticides that have recently been used in and around the home's premises were found to leave residues. Residues of many pesticides were found in and around the home even when there was no known use of them on the premises. chlordane termiticide treatments did not show evidence of chlordane in the indoor environment.

	The representative pesticide classes that were found in the indoor environment include the banned or canceled long lasting chlorinated cyclodienes that were widely used in termite control and are considered as persistent organic pollutants (POPs). These POP termiticides no longer in use include chlordane, heptachlor, aldrin, endrin, and dieldrin. Dieldrin is very toxic and extremely persistent. In addition, chlordane's breakdown product oxychlordane and other components such as octachlor and nonachlor are commonly found. Heptachlor, and its toxicologically active breakdown product heptachlor epoxide are all commonly found in older homes with termiticide treatments, Chlorinated 4,4'-DDT and degradation products DDD and DDE were heavily used both agriculturally and domestically for insect control. These pesticides can still be found in homes although they are no longer used. These pesticides are considered as persistent organic pollutants (POPs) that are all under current international negotiations. Dacthal - a common lawn and turf herbicide, atrazine - an outdoor and high agricultural use herbicide, folpet (fungicide) were also found. The toxic organophosphate insecticides that are found include moderately persistent chlorpyrifos with high indoor, outdoor, and termiticide use, dichlorvos (DDVP) diazinon, and malathion. Other commonly used insecticides that are commonly found include the moderately toxic pyrethroids such as cis and trans-permethrin, Pentachlorophenol (PCP), and the cleaning component o-phenylphenol. The toxic N-methylcarbamate bendiocarb and propoxur are not considered to be persistent, yet are commonly found in indoor air and dust. Older homes (e.g., built in 1930-40s) were found to have high levels of indoor pesticides. Homes built in the 1960s showed second highest levels. Canceled pesticides such as chlordane, aldrin, and dieldrin were common termiticides in the 1970s but, have not been since 1988. Homes from the 1980s that did not have sub-surface or preconstruction
Population(s)/ecosystem(s) exposed statewide	Any NJ person inhabiting a residence or location that has had indoor chemical pesticide treatments will be exposed. To a variable extent, outdoor treatments will also contribute to indoor pesticide exposure. Conventional termiticide treatments have been and will continue to be performed in NJ adding up to some pesticide exposure.

Quantification of exposure levels statewide, including populations at significantly increased exposure **(include indoor air as separate category as appropriate)**	Currently, it is not possible to track the number of indoor pesticide applications or number of termiticide treatments, nor the number of people or households exposed each year. The NJDEP Pesticide Control Program (PCP) does have the legal authority to request use records from professional applicators. PCP carried out a use survey to estimate termiticide use in 1992 and indoor chemical use in 1993. These estimates do NOT include the amounts of generic pesticides used by homeowners. Also not tracked by NJ are sales of generic pesticides that are directly available for purchase and use by the public "over-the-counter." To get an idea of the number of households or premises that may be effected by termiticide treatments on a yearly basis in NJ, an estimate of the total amount of termiticides used in NJ may be divided by a hypothetical "average" amount used in a conventional chemical termiticide treatment. In the last termiticide use survey performed by NJDEP/PCP, there were six different chemicals available for termiticide use and the reported total used was almost 141,000 pounds in one year. Assuming an average whole-house treatment uses about 8 to 16 pounds of termiticide active ingredient, with an estimate of 9,000-18,000 residences or buildings treated in a single year. A single organophosphate pesticide, chlorpyrifos, comprised about 55% of the total pounds of termiticide amounts reported as used. The second most used termiticide was the organophosphate isofenphos contributing another 27%. Isofenphos is no longer allowed to be used as a termiticide.

Specific population(s) at increased risk	A recent development is the use of termiticide bait stations. Any reduction in health risks resulting from their use relative to conventional chemical termiticide use in NJ has not been calculated. The New Jersey Poison Information and Education System (NJPIES) serves as the Poison Control Center for NJ. NJPIES received more than 4500 pesticide related calls in a year, of which 82% originated from a residence. For the pesticide-related calls, ingestion was the most common route of exposure (61%) followed by inhalation (20%). In the future, these data and additionally needed residential information may provide useful indicators of potential residential pesticide exposure and hazards. The specific sub-populations at risk are infants and children. This group is cited as particularly exposed and susceptible to pesticides from tracked-in dusts. The percentage of NJ children that are involved in common household accidents and poisoning involving pesticides are unknown. Asthmatics, severely allergic and hypersensitive individuals are also at increased risk. Additionally, asthma and allergic responses may be aggravated by solvents and other non-pesticidal substances included in the product. Increasing use of home, lawn and garden pesticide applications in suburban areas increases the potential exposure for this sub-group. Another sub-group at risk consists of urban pesticide users that obtain their pest control illegally (and more cheaply) from local neighborhood distributors. There are State and Federal investigations into the illegal home use of highly toxic agricultural pesticides such as methyl-parathion, another organophosphate. This initiative needs to be more fully implemented in NJ. Homes on or in pesticide-applied agricultural areas are another subgroup that would be expected to have higher elevations of pesticides residues indoor, both in the indoor air and dust from track-in.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) **(include indoor air as separate category as appropriate)**	The exposure level to specific sub-populations in NJ cannot be directly quantified. The specific amount of each pesticide for each sub-population that is potentially exposed is not available.
Dose/Impact-Response Assessment	

Quantitative dose/impact-assessment employed for each population considered	The extensive and in-depth risk assessments for organophosphates are available on the EPA website and are continually being updated. Ultimately 40 OPs will be assessed. Most of these actions are being triggered by the 1996 FQPA regulation that specifically considers risk to children and infants. On the national scale, EPA started to formally re-review the risks-benefits of organophosphates. Isofenphos is already not allowed to be used as a termiticide. Chlorpyrifos - the highest homeowner used organophosphate termiticide and insecticide has been determined to be unsafe for most homeowner use and especially for children. The manufacturer was urged to withdraw it from all home and garden uses. (EPA May, 2000). The registrant has not agreed to this as of December 2000. Another organophosphate of major use in NJ undergoing major review and assessment is diazinon. In December, 2000, EPA obtained an agreement that eliminates all indoor uses of diazinon effective March 2001, and starts a phase-out of lawn and garden uses yielding at least a 75% reduction in diazinon's use nationwide.
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	Estimated numbers of accidental pesticide poisonings for adults and children are unknown for NJ. Common pesticide incidents involve dermal contact by accidental spillage, accidental ingestion, inhalation, ocular irritation or damage, etc. Funding and research for NJPIES may provide information and data to estimate number and type of occurrences from calls to the NJ poison control center.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	EPA has concerns about long-term health effects of the chlorinated cyclodienes including effects on the liver and the Central Nervous System (CNS), and causing an increase in the risk of cancer. Persistent pesticides are still being found many years after their use stopped and may drift global distances from application points (e.g., Canada, arctic).
Size of population(s) affected	**High** - On a national scale, 75% of US households used at least one pesticide indoors during the past year. Most of NJ residents are expected to be exposed to some level of pesticides in their indoor environment. This may be directly through use, or indirectly in their workplace, school, store, or marketplace. Data gaps exist, and it is unknown how many NJ residents are directly exposed and what percentage of these are affected by the indoor pesticide residue exposure.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	**High**. The uncertainties in the assessments are high mainly because of the wide spread data gaps for both the exposure data of susceptible subgroups to many pesticides in various indoor environments and the lack of data in the assessments of resultant and cumulative risks for so many pesticides. There is insufficient data and knowledge of the impacts of pesticides; what concentrations of which pesticides and formulation solvents and "inert ingredients" are necessary to produce the pesticide impacts.

	Much of the pesticide impact data is based on data obtained from toxicological testing utilizing high doses of the pesticide in animals. EPA among others has serious concerns about the chronic impacts of low doses especially on the endocrine system and reproduction, the neurological and immune systems, cognitive and behavioral systems such as learning, and memory.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	**High** - There has been a high level of changes based on recent re-assessments. Additional data may be expected to uncover additional subpopulations at risk, Future risk re-assessments should take into consideration the decrease in risk caused by withdrawal or cancellation of high risk pesticides, and the new risks posed by the use of substitutes to control pests. For NJ, there is a large potential for reducing risk in the area of pesticides in the indoor environment through funding research for indoor air pesticide use, air monitoring, funding assessment, development, and implementation of best management practices, increasing alternatives to chemical pesticides, and public outreach.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	**0.** There is potential for some future changes in certain areas because under FQPA 1996 there is a national impetus for examination and reduction of high-risk pesticides. It is unknown at this time whether the new federal and state administrations and legislative bodies will support further pesticide risk reduction. Pesticide manufacturers will fill the market niche with new pesticides and substitutes. The questions remain as to whether pest control will be achieved with a true reduction in risk or whether pesticide substitution will create different risks and negative impacts.
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	**High.** There would be a high impact resulting from a catastrophic event involving highly toxic pesticide use or misuse in an indoor setting. There is also a potential for accidental poisonings especially for children and infants through ingestion of toxic household pesticides. There are also substantial impacts possible in a communal residence, workplace, or school. The likelihood of such a catastrophe is low.
Extent to which risks are currently reduced through in-place regulations and controls	**Moderate**. Reductions in risks from in-place regulations are based on existing federal regulations, product labeling and national data. NJ is a densely populated state with enough varied pests and indoor pesticide use to warrant generating and updating exposure data and re-assessments. This will allow targeting higher sources of risks for efficient control and risk reduction. State regulations do not have specific regulations for pesticide storage in stores and marketplaces.

Issue: Pesticides-Indoor
Author: Ateta Wowk
Version: 02/01

Relative Contributions of Sources to Risk (H,M,L)	.
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	**L** (pest control practices that increase indoor pesticide residues and thereby exposure)
Small business industry	**L** (same as above)
Transportation	**M** (pest control, disinfection, fumigation etc., in airplanes, buses, and mass transit vehicles and stations.
Residential	**H** (exposure from both residential indoor and outdoor applications, domestic pesticide storage. Also includes place of work, school, indoor marketplaces and stores, especially where pesticidal products are sold).
Agriculture	**M** (function of proximity to pesticide applications to agricultural areas)
Recreation	**M** (localized exposure possible near golf courses and recreational grass playing fields)
Resource extraction	NA
Government	**L** (gov. pest control activities affect outdoor environment more than indoor)
Natural sources	NA
Contaminated sites	**L** (localized exposure from old termiticide application areas or old agricultural spill / dump sites)
Diffuse and non-NJ sources	NA
Sediment	NA
Soil	**H** (pesticides in soil, dirt, and particulates)
Non-local air sources (including deposition)	**M** (long range drift or global transport)
Biota sinks	**L** (low impact from indoor pesticide use)

Human Health Issue Summary: Pesticides -Indoor

What is it? Pesticides are used indoors largely to control pests. While risks from pesticides used in agriculture or other outdoor applications may be significant, EPA estimates that indoor exposures may account for 80-90% of total exposure. Dozens of pesticides have been identified in indoor air.

Who's at risk? Households that use pesticides for insect and rodent control are the primary focus of indoor pesticide risks. Children may be particularly at risk. As with other

Issue: Pesticides-Indoor
Author: Ateta Wowk
Version: 02/01
pesticide health effects, the wide range of pesticides in use makes it difficult to pinpoint individual health endpoints. However, chronic pesticide health effects include nervous system damage, immune, developmental effects. Acute effects may include headache, dizziness, dermal, respiratory and eye irritation..

What is the extent of human health problem in New Jersey? While the number of accidental poisonings is not precisely known, it is known that 4,500 pesticide-related calls were made to the New Jersey Poison Control Center. 82% of these calls originated from a residence. There is little information on chronic risks and it is difficult if not impossible to separate effects of indoor exposure from food, water, and outdoor exposures.

What's being done Federal regulations require labeling and in some cases, the registration for certain uses is deleted decreasing exposures. The potential for New Jersey research and regulation may reduce risks significantly.

Risk Summary
Indoor exposures to pesticides are probably greater than outdoor exposures or exposure through food and water ingestion. The Technical Work Group ranked the issue Medium.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	**Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)**	**Are there discreter communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)**	**Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)**
M (4)	M (4)	Y (5)	M (4)

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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor Description of stressor (including etiology)	**Pesticides - Outdoor** Pesticides are any substances or mixture of substances employed to destroy, prevent or control pests. By design, the very nature of pesticides creates some risk of harm to humans, animals, plants, living organisms, or environment. Approximately 600 registered unique pesticidal substances are unified only by this characteristic. These substances have greatly different chemical, physical, and toxicological (acute and chronic) characteristics, differing environmental fate and transport characteristics that determines movement and persistence. Pesticides may be commonly classified into broad groups according to the pest to be controlled. For example, insecticides control insects, herbicides control plants and weeds, fungicides control molds or fungal growth fungicides. There are also pesticides that are insect- or plant- growth regulators, and antimicrobials. Pesticides also can be classified on the basis of general chemical characteristics. The broad chemical pesticide classes includes halogenated organics (DDT, methoxychlor, methyl bromide), chlorinated cyclodienes (dieldrin, and others banned from use), inorganics (sulfur), arsenicals, metals (copper containing substances), organophosphates (OPs), pyrethroids, carbamates, phenoxy-herbicides acids and esters, chlorinated fumigants (methyl bromide, dichloropropane), chloroacetanilides, triazines, substituted ureas. Other classifications may include microbiologically based (Bt endotoxin) or biologically derived substances (pheromones, growth regulators, and botanical oils). Petroleum hydrocarbons, solvents, surfactants are also contained in the formulations or listed as "inerts." Pesticides of a similar class may be associated with certain symptoms or a common mechanism of action. Human pesticide exposure can result from the direct use of pesticides in the outdoor environment. A major source of this type of exposure results from the widespread county government, extensive commercial and homeowner use of herbicides, insecticides, and fungicides for pest control. The sites of exposure include areas of pesticide use on residential lawns, turf, home and outdoor gardens, parks, camp-sites, playgrounds, driveways, roads and rights-of-ways, and golf courses. Exposure is growing through increased use of pesticides in various outdoor settings, such as areas controlled for gypsy moths, black flies; and disease vectors such as ticks, fleas, and mosquitoes. Another important source of exposure to humans is the indirect exposure through drift, evaporation and atmospheric transport by air, mist, fog, and rain. Significant amounts of pesticide exposure can result from tracking pesticides from the outside environment into homes or the indoor environment. Pesticides in the atmosphere or outdoor air environment are associated with areas of highest outdoor pesticide use. Pesticides associated with agricultural applications may be found to be transported long distances.

Stressor-specific impacts considered including key impacts	Low levels of long-lived or persistent pesticides are present in the atmosphere throughout the year. These pesticides have been found wherever they are studied. Some of these persistent pesticides have the ability to bioaccumulate in humans and in the food chain. Historical Use Pesticides are those pesticides that include the banned or canceled long lasting chlorinated cyclodienes and DDT- type pesticides that were widely and heavily used in agriculture and household soil and termiticide treatments. Many of these pesticides are internationally known as persistent organic pollutants (POPs). The POPs no longer in use include chlordane, heptachlor, aldrin, endrin, and dieldrin. Dieldrin is very toxic and extremely persistent and all uses have been cancelled since 1987. Chlordane and heptachlor have had all uses canceled since 1988. Chlordane and its breakdown product oxychlordane; and other associated components such as octachlor and nonachlor are commonly found. Heptachlor, and its toxicologically active breakdown product heptachlor epoxide are also commonly found. Chlorinated 4,4'-DDT and degradation products DDD and DDE are also widely found in the NJ environment. Outdoor herbicides are widely used for lawn care, turf, rights-of way, and golf courses in NJ. There are hundreds of different pesticides that may be used for controlling the varied types of insects, weeds, and fungi pests found outdoors. Common pesticides used in lawncare include herbicides such as pendimethalin and glyphosate, and benfluralin. The acid herbicides such as the phenoxy and chloronicotinyl type of herbicides such as 2,4-D, dicamba, MCPA, mecoprop, clopyralid, and triclopyr make up a large percentage of the turf and lawncare use herbicides. The organophosphates such chlorpyrifos, trichlorfon, make up a large fraction of the Insecticides used for lawncare, turf, and golf courses. Golf courses account for a relatively intense use of pesticides on small land areas. A single fungicide such as chlorothalonil makes up the largest proportion (>40%) of the pesticide use on NJ golf courses. Of the over 600 pesticides currently in use, relatively few pesticides are commonly used for mosquito, black fly, tick, and gypsy moth control. These pesticides can be broadly classified into categories of oils, surfactants, biological pesticides, pyrethroids, carbamates, and organophosphates. Pyrethroid-type pesticides are widely used for insect control including mosquitoes. Organophosphates such as malathion have been heavily used to control adult mosquitoes. Petroleum hydrocarbons in the form of oil and biological pesticides such as Bacillus Thuringiensis (Bt) are widely used for mosquito control in the larval stages. Bt is extensively used by NJ government agencies for achieving control of black flies and gypsy moths. Commercial applicators use the carbamate, carbaryl for the non-larval stage control of gypsy moths. Products containing carbaryl and the pyrethroid permethrin are widely applied for tick control in NJ. Surfactants, such as polyethoxylated alcohols (POE), are also being used to control mosquito and midge larvae and pupae by creating a surface film on water. Certain chemically related pesticides may have a common mechanism of toxicity such as the organo-phosphates (OPs). The hazards of 40 individual OPs are currently being comprehensively assessed on a case by case basis (EPA, NTP). Upon exposure, all OPs are neurotoxic, causing cholinesterase inhibition and related clinical symptoms, up to and including death. (EPA & HIARC 1997,1998). All are considered highly poisonous, except for acephate and malathion which are classified as moderately poisonous. Only a few OPs (e.g., parathion, phosmet) are classified as possible human carcinogens, or as causing reproductive effects (e.g., trichlorfon, guthion/azinphos-methyl). OPs are not considered to accumulate and persist in the body. OPs may cause neuropathology of the visual system or effects on cognitive functions such as learning and memory.

	Many organophosphates (OPs) and carbamates are ranked as highly acutely toxic pesticides that can be inhaled, ingested or absorbed through the skin. Acute symptoms of OP poisoning develop within minutes to hours after exposure with symptoms first appearing in the respiratory system. Since the enzyme acetylcholinesterase is inhibited, the symptoms are related to effects on the central nervous system, muscarinic, and nicotinic receptors. Enzyme depression, in the case of plasma pseudo-cholinesterase, generally persists for several days to a few weeks; red blood cell enzyme activity may remain depressed for 1-3 months after over-exposure. The effects of over-exposure include nausea, headaches, vomiting, diarrhea, and weakness. Malathion itself is one of the few OPs having a low acute mammalian toxicity and low persistence especially in water. However there are several breakdown products with higher toxicity than the parent malathion. Temephos is an organophosphate with low toxicity to mammals. It may bioaccumulate. It is of low persistence in water, low to moderate persistence in soil, and high in plants. Repeated low dose exposure in animal experiments (gestating rats) with another OP - chlorpyrifos - indicated extensive neurochemical and neurobehavioral changes. As a result of human neurotoxicological data and new data indicating that OPs may affect fetal brain development, EPA increased the applicable safety factor for women and children by 3.3 times for chlorpyrifos. To decrease the new allowable margin of exposure, the manufacturers agreed to decrease allowable chlorpyrifos uses. The agreement will result in decreasing residential household and yard use. Chlorpyrifos is a moderately persistent OP with 50% of its use nationally classified as residential. Sale of chlorpyrifos for residential use will stop on December 2002 and lawn, garden, and turf uses end on August 2003. Diazinon, is an OP with low to moderate persistence. It is considered the most widely used pesticide by homeowners on lawns. Use was diminished beginning on March 2001, when all outdoor uses began phase-out, eliminating about 75% of the use by 2003. Acidic herbicides such as the phenoxy acids are found to persist in the environment, having short to moderate half-lives, ranging from less than 2 weeks to 1-2 months. Many of the characteristic properties of the pesticides are modulated by being formulated into salts and esters. The pesticide triclopyr acid is moderately persistent and is also mobile in the environment. There is concern here on ecological impacts as well. Oils, petroleum hydrocarbons, are extensively used for mosquito larva control. Solvents such as petroleum hydrocarbons are commonly used to aid in solvating the pesticides that are not very water soluble. They are considered to be “inert” yet their human health impacts need to be considered, as the levels may temporarily exceed adverse effect levels. This is especially important for the populations with respiratory and immune system challenges. Methoprene is an insect growth regulator and is a long chain hydrocarbon ester. The toxic impact of hydrocarbons on human health is low; however, respiratory stress for sensitive exposed subpopulations needs to be considered. The effects of synergists and inerts need to be evaluated as to their adverse effects. Synergists are substances added to pesticide formulations to increase the toxicity of the product. Synergists, such as PBO and MGK 264, may be added to pesticides such as pyrethroids or carbamates to increase the toxicity by interfering with their detoxification. One of the target organs for side effects of PBO is the liver. Some animal studies indicated liver tumors at high doses. Inerts are non-pesticidal ingredients that are included in the product formulations and used as carriers, solvents, and stabilizers. Solvent used in pyrethroid products include petroleum hydrocarbons. system.

Exposure Assessment	The effects of phenoxy acidic herbicides fall into the slight to low toxicity. However, many of these product formulations may be corrosive and cause severe eye irritation and even permanent eye damage. Very severe overexposure is characterized by effects on the liver, kidney, thymus, and spleen tissue. Clinical manifestations of overexposure involve twitching, muscle spasms, drooling, slow heart beat, low blood pressure, and unconsciousness. In farmers, muscle weakness, anemia, and digestive problems were observed. Some pesticides of this class were observed to be weakly mutagenic and to cause reproductive effects. However, with normal use and exposures, these effects are considered unlikely.
Exposure routes and pathways considered (include indoor air appropriate)	Pesticidal strains of the bacteria Bacillus Thuringiensis (Bt) are used to control black flies, mosquitoes, and gypsy moths larval stage. The varieties of Bt used as pesticides in the outdoor environment do not present a human hazard. However, it is recommended that Bt be linked to the NJCRP Ecological Technical Work Group report considering the ecological long term effects of pesticidal use and issues of resistance. Synthetic Pyrethroids are neuropoisons, affecting the sodium channel in the nerve membrane, nerve impulse generation and conduction. Pyrethroids, mostly used as insecticides, are classified into four types. Type I includes moderately toxic permethrin. Permethrin's toxicity also depends on the composition of the formulation used. Mammalian poisoning is characterized by tremors, hyperexcitation, ataxia, convulsions, and paralysis (T syndrome). Permethrin and bifenthrin are considered to be weak carcinogens. The mode of poisoning from Type II pyrethroids that contain an alpha-cyano group is different form Type I. Type II exhibits more open time for the affected nerve membrane sodium channels, and is characterized by hypersalivation, hypersensitivity to stimuli, choreo-athetosis and C-syndrome paralysis. Pyrethroid exposure may be accompanied by tingling sensations of the skin - often in the facial areas. The Type II pesticides containing cyano groups have been suspected of causing seizures or parasthetic effects. These synthetic pyrethroids are considered to be moderately persistent. Type III pyrethroids are represented by slightly toxic resmethrin. The pyrethroid d-phenothrin (sumithrin) exhibits low toxicity and relatively rapid degradation rates. All pyrethroids may be absorbed through the skin, so direct dermal contact should be avoided as well as inhalation and oral exposure. Most pyrethroids are considered to be moderately to highly irritating to the eyes causing stinging, tearing, and blurred vision. At high doses, symptoms such as a stuffy runny nose, coughing, headache, fever, irregular heartbeat, nausea, weakness, and tremors may be experienced. Synthetic pyrethroids have been the alternative to the banned pesticides such as DDT and exhibit similar toxicological mechanisms to those of DDT. However, they are far less persistent and more degradable. Overall, pyrethroids are more toxic to insects than to mammals and are not associated with chronic neurological impairment. From high dose animal studies, adverse effects include reproductive, immune and neurological effects. Developmental effects such as neuro-receptor alteration leading to hypersensitivity, reduction in neural signal transmission efficiency and behavioral abnormalities may be seen in prenatal and neonatal nervous systems. Disturbances in the endocrine system, lymphocyte function, and thymus weight may contribute to immune response suppression. Chronic exposure with resmethrin in animals indicated effects on the thyroid. Pyrethroids may act as environmental estrogens contributing to reproductive dysfunction and developmental impairment. Future studies should include transgenerational effects of pyrethroids.
	To address a national concern, evaluating endocrine disruption effects is one of the newest EPA initiatives to be established under the Endocrine Disruptor and Screening Program (EPA Report to Congress 2000). Most pesticides have not been fully

	evaluated with respect to endocrine disruption. EPA tests and criteria are still being developed (ES&T, October 2000) and have been criticized as being deficient in the ability to define and assess low dose effects of pesticides on the immune system and neurological structure (NTP Panel 2000). Most pesticide testing, evaluations, and assessments are heavily based on or derived from high dose animal testing data. Vinclozolin and its metabolite is being reassessed and curtailed because of unreasonable risks to the endocrine of the heavily used organophosphate pesticide chlorpyrifos and diazinon. The pathways of concern include: outdoor pesticide application, ground spraying, and aerial application. There will be some unintentional exposure through drift. Some of the pesticides will be inhaled, some fraction will be absorbed through the skin through dermal contact. Some of the vapor or mist will contact mucosal membranes in the nose or eyes. The environmental fate may include some incorporation into soil and sediment, or runoff or water, and uptake into biota and food chain. Exposure may result from residues carried by soil particulates and dusts. Significant amounts of outdoor pesticides enter the indoor environment through track-in. This results in higher levels in indoor dust than outdoor soil concentration levels. These levels were of enough risk and concern on a national level that EPA moved to negotiate severe cutbacks on the residential home and garden use
Population(s)/ecosystem(s) exposed statewide Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	Data are not being collected and tracked to be able to assess specific area/acreage or populations to pesticide exposure. Since mosquito control programs are likely to be expanded, this data gap still needs to be addressed. A large percentage of NJ households have used had some kind of indoor pest control. Many households use lawn or garden pest control services, some perform their own pesticide treatments. The NJDEP/Pesticide Control Program performed a survey of the amount of pesticides applied by licensed professionals for mosquito control in 1998. This survey does not include any pesticide use performed by private citizens. The survey found that a total of over 63 thousand pounds of pesticide were applied for mosquito control in NJ in 1998. This was before the West Nile-like Virus outbreak of 1999 that was accompanied by increased mosquito control. There will be a new survey taken to fill this data gap. The largest use of pesticide was oil (73%). Oil containing petroleum hydrocarbons can increase air volatiles and ground level ozone and water bodies are affected when the oil is spread as a larvicide. The next highest use pesticides are the organophosphates malathion (14%) and temephos (6%). While the last survey of 1998 did not indicate a high use of the synthetic pyrethroids, it is anticipated that their use will increase significantly in future NJ mosquito control efforts. The results of mosquito pesticide use surveys for the years 1999 and 2000 were available in Spring 2001. Amounts of pesticides applied to lawns and turf by commercial applicators are tracked by the NJDEP/Pesticide Control Program. Over 518,000 pounds of pesticides were commercially applied in 1998. Of that amount used, 65% were herbicides. Phenoxy acid herbicides such as 2,4-D and mecoprop, and the dinitroaniline herbicide pendimethalin are widely used. About 42% of the insecticides used were represented by two OPs - chlorpyrifos and trichlorfon. A large data gap is the public's use of "over-the counter" pesticides bought in stores.
	According to data from the NJ Department of Agriculture, Gypsy moth damage has been recorded over 3 major cycles in the past 30 years. In 1997, over 4400 acres were treated with Bt as part of NJDA biological suppression program. In 1998, the

	acreage treated fell to 760 acres. Aerial surveys indicate increases in infested acreage in 1998. Biological suppression decreases the demand for synthetic pesticide use that may carry higher risks. Outdoor playgrounds, parks and campgrounds are also treated for pest control. The exposure from this source is unknown. Quantification of exposure levels is not currently possible, and this identifies a serious data gap. Specific programs and research have not been established to address the data gap in the numbers of the general population exposed, of any identified vulnerable subgroup, and the exposure levels. Also, data on specific areas or acreage, or numbers of people exposed in each county are not available.
Specific population(s) at increased risk	There are statewide sub-populations of significant size that are exposed and considered to be at higher risk. Subgroups may include elderly (those over 65 years old), infants, and children (12 years and under), females/males of reproductive age (13 years +), and women that are child-bearing or nursing. Young children due to frequent mouthing or hand to mouth behavior, are at higher risk of daily exposure to pesticides from exterior domestic applications that may exceed levels of concern. This has been shown for diazinon, while the levels are just slightly lower for chlorpyrifos. Data gathered from studies indicate further investigations the relationship between pesticide use and pregnancy and fetal effects of pesticides. An increased risk of brain tumors in young children was observed in one study only for flea and tick products-especially sprays and foggers. There are a number of pesticides used, chlorpyrifos is one of the used OPs. Sensitive individuals such as those with chronic lung disease (asthma; etc.), CNS dysfunction, sensitivity to specific pesticides or formulations could experience adverse effects or an increase in their symptoms. There may be increased effects for people with sensitive or compromised immune functions.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	More research is needed to address the data gaps that make it impossible to quantify the exposure levels and impacts of that exposure for the identified vulnerable subgroups in NJ. It's expected that the actual levels of exposure would be similar to that of exposure levels found for that source for the general population. However, the assessment of the impacts of the exposure of the mixtures of pesticides and related products presents an enormous challenge.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	Data Gap. Assessment for each population in NJ is currently not available for all of these pesticides.
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences	Data Gap. There currently is no metric to assess the number of cases/occurrences in NJ. Currently in NJ, there is no coordinated way of tracking incidences of pesticide poisonings. The medical system also does not

(specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	coordinate tracking emergency room visits related to pesticide poisonings or over-exposures. The New Jersey Poison Information and Education System (NJPIES) serves as the Poison Control Center for NJ. However, incoming calls are tracked and NJPIES received more than 4500 pesticide related calls in a year, of which 82% originated from a residence. For the pesticide-related calls, ingestion was the most common route of exposure (61%) followed by inhalation (20%). In the future, these data and needed residential information may provide useful indicators of potential residential pesticide exposure and hazards.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Data Gap. No metric currently exists to be able to assess the frequency and severity of effects.
Size of population(s) affected	High -Many, if not most, NJ residents will be affected to a certain degree. NJ residents applying or indirectly being exposed to pesticides in the outdoor environment such as disease vector control, lawn care or garden, golf course, or proximity to agricultural applications; and other outdoor pest control operations will make up the population that is impacted.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	High uncertainty. The existence of serious data gaps currently makes this assessment impossible to complete . Many of the pesticides applied are not being monitored. There is insufficient data and knowledge of the impacts of pesticides; concentrations of pesticides and formulation solvents and "inert ingredients" are necessary to predict the pesticide impacts. The uncertainties in the assessments are high mainly because of the wide spread data gaps for both the exposure data of susceptible subgroups to many pesticides in various indoor environments and the lack of data in the assessments of resultant and cumulative risks for so many pesticides. Much of the pesticide impact data is based on data obtained from toxicological testing utilizing high doses of the pesticide in animals. EPA among others has serious concerns about the chronic impacts of low doses on especially the endocrine system and reproduction, the neurological and immune systems, cognitive and behavioral systems such as learning, and memory.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	High potential exists for additional data to result in significant change. Exposure data in needed and this data could be obtained. More difficult is a full impact assessment that is also necessary for formulating a reasonable risk estimate. There has been a high level of changes based on recent re-assessments, e.g., reduction of home uses of chlorpyrifos and diazinon. Additional data may be expected to uncover additional subpopulations at risk. Future risk re-assessments should take into consideration the decrease in risk caused by withdrawal or cancellation of high risk pesticides, and the new risks posed by the use of substitutes to control pests. For NJ, there is a large potential for reducing risk in pesticides in the outdoor environment through funding research for monitoring, assessment, development and implementation of improved pest and pesticide management practices, increasing alternatives to chemical pesticides, and public outreach.
Potential for future changes in the underlying risk from this stressor	+++ There is a good potential for changes in the underlying risk from using chemical pesticides for outdoor vector control. With increasing data, research, best management practice development and outreach, reasonable vector control while minimizing risk

(+++, ++, +, 0, -, =, where + is improvement)	can be achieved. Since many of the pesticides are toxic to insects, aquatic macroinvertebrates, and fish, these pesticides that are human health stressors should also be linked to the assessment of the *Ecological Technical Work Group.*
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	Medium-High potential impact. Catastrophes could result from accidents, misuse, spills during pesticide transport, or application, storage, floods, etc. The severity of the impact is dependent on the particular pesticide involved.
Extent to which risks are currently reduced through in-place regulations and controls	Medium The current risks are controlled mainly by pesticide labels and federal (EPA) registration, and the licensing, certification, and permitting functions of NJDEP/ Pesticide Control Program. Not addressing the data gaps along with the potential of increasing outdoor pest control is likely to increase risks. There are many opportunities for reducing the risks to so many pesticides being used - however, the varied number and site of pesticide use makes
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	N/A
Small business industry	N/A
Transportation	Medium- Pesticides used on rights-of way, parking lots, roadsides, railroad tracks, power line areas, etc. Over 54,000 pounds of pesticide were applied in a single year. While this does not represent a high risk of a direct threat to public health, there may be an indirect threat in certain areas of vulnerable ground or surface water. Ecological effects need to be addressed.
Residential	High- High impact. Lawn and garden, vector control (e.g., tick) pest control has high direct and indirect impacts on human health.
Agriculture	N/A
Recreation	High- Golf courses use heavy amounts of pesticides over a relatively small area. Fungicide use such as chlorothalonil which by itself accounts of over 40% of the total pesticides used.
Resource extraction	N/A
Government	Medium- Currently medium risk but increasing for specific areas especially areas, under county mosquito pest control.

Natural sources	N/A
Contaminated sites	N/A
Diffuse and non-NJ sources	
Sediment	Unknown
Soil	Unknown
Non-local air sources (including deposition)	Unknown for NJ. There is data indicating that long distance transport and deposition of pesticides occurs through the atmospheric matrices of air, rain, snow, fog, and aerosols. (USGS, 1995; Majewski and Capel, 1995). Risk may increase due to increase in aerial spraying for mosquito control. Agricultural aerial applications linked to pesticides found in air, and with lower concentrations found in rain. Historical use pesticides such as DDT, chlordane, etc., are still being found due to long range air transport and deposition. Many pesticides that are used are not monitored.
Biota sinks	N/A

Human Health Issue Summary: Pesticides - Outdoor

What is it? There are more than 600 pesticides in use in New Jersey. They are used to control unwanted plants (herbicides), insect pests (insecticides) and fungus (fungicides). They are designed to be toxic to target species, and in most cases create risk to humans as well. The application of pesticides is used in agricultural settings, in areas adjacent to human settlement to reduce the risk from insects as vectors of disease, and in residential areas as a convenience for growing home gardens and lawns and reducing insects in the home. For this problem description, the focus is on exposures from outside applications and does not include contamination of drinking water or food.

Who's at risk? Exposure to humans takes place primarily during application, or dermal contact in areas after application. Exposure can also occur secondarily as a result of atmospheric drift and evaporation. Therefore, some of the greatest risks are place and time specific. There are chronic effects as well, but for non-occupational exposures, these are more probably the result of food water and indoor exposure addressed elsewhere. Persistent pesticides such as DDT and chlordane can bioaccumulate. For such pesticides, secondary exposure pathways including the food chain are important. The specific effects from pesticides are as varied as the chemistry of the compounds. In some cases, effects are neurotoxicity, in others, nausea and diarrhea, in others immune system responses and in still others, cancer and endocrine disruption.

What is the extent of human health problem in New Jersey? In general, the lack of data about human exposure and body burdens makes quantifying the effects from pesticide exposure difficult. The main evidence cited in technical assessments for health effects from outdoor exposures is the frequency of calls to poison hotlines for pesticide-related questions and concerns. The New Jersey Poison Information and Education System receives about 4,500 calls per year related to pesticides and almost 1,000 of these may be related to outdoor exposures.

What's being done? Commercial pesticide applications are regulated to minimize chronic exposures and training of applicators is intended to reduce acute exposures. Most persistent pesticides are banned from use.

Risk Summary

Issue: Pesticides Outdoor
Author: Areta Wowk
Version: 05/01
The range of possible outcomes from pesticide exposures combined with the quantities of pesticides applied in New Jersey led the Technical Working Group to assign a Medium ranking of health risks.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
MEDIUM (3)	High (5)	H(4)	MEDIUM/High (4)

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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Pesticides -Water**
Description of stressor (including etiology)	Pesticides are widely used in NJ in moderate to heavy quantities for a variety of purposes including agriculture, structural pest control, gardening, landscaping, turf, lawncare, golf-course and right-of-way maintenance. There are hundreds of different pesticidal substances that may be used for controlling the varied types of insects, weeds, and fungi pests found in NJ. There are many chemical classes of pesticides that are found in NJ waters, including but not limited to organochlorine, organophosphates, carbamates, pyrethroids, triazines, chloracetanilides, and inorganics There is a growing concern among New Jersey communities about pesticide presence in surface- and ground- water systems and the possible adverse effect they may have on human health and the aquatic ecosystem. Pesticides of every major chemical class used may be detected in New Jersey's ground and surface waters. NJ waters are particularly vulnerable because of conditions favorable to contamination by pesticides. There is widespread use and application of pesticides that have significant potential to contaminate by leaching into ground water and running-off into surface water under certain conditions. NJ has much available ground water and surface water, such as streams, rivers, and lakes, that are hydrogeologically sensitive or vulnerable to contamination. NJ has significant rainfall that promotes this movement of pesticides into both ground water and surface water. There are areas where ground and surface water are connected. In NJ, ground water commonly provides most of the base-flow source for surface water. Pesticides are commonly present in low concentrations in shallow ground water beneath agricultural areas. Ground water pesticide concentration levels may show seasonal variability and maximum levels following springtime applications. Pesticide levels in surface water follow an even stronger seasonal pattern following pesticide application and the presence of runoff conditions. Based on national studies, factors most strongly associated with pesticides in both ground water and surface water are high pesticide use, high levels of precipitation or irrigation, and shallow, inadequate or older wells. Surface water contamination is strongly dependent on the amount, pesticide type, and timing of pesticide application. Pesticides can also contaminate water by spills, leaks, and improper disposal.

	The drinking water for about half of NJ residents in or near urban areas is obtained from surface water (streams, reservoirs). Approximately another half of the population is served by drinking water obtained from ground water. In rural areas of southern NJ, much of the drinking water is obtained through wells that tap into ground water. The main concern here is that pesticides in ground and surface water used for drinking water may endanger human health. Periods of exceedances related to seasonal pesticide use have been found. Another concern is the effects on aquatic ecosystems. This information is linked to the pesticide report from the Ecological Work Group. Pesticides contaminating ground water are difficult to remove or clean up, while pesticides in surface waters increase treatment costs to produce suitable drinking water. There are only about 26 federal Maximum Contaminant Levels for pesticides in public water drinking water supplies. Of these, 8 are "historical use" or pesticides no longer used in NJ. Many of these canceled pesticides also are listed as persistent organic pollutants (POPs). The POPs are considered to have such serious, wide-spread and long-term effects that they are subject to international agreement. POPs such as DDT, DDD, DDE, aldrin, and dieldrin do not have an enforceable drinking water Maximum Contaminant Level (MCL). Some of these "historical use" and POPs pesticides are often found in both ground water and surface water. The most widely used pesticides in NJ agriculture belong to triazine and chloracetanalide type of herbicides. The major triazine pesticides include atrazine, cyanazine, and simazine. The major chloracetanilides include alachlor, metolachlor, and acetochlor. These herbicides are able to both dissolve in water and adsorb onto soil particles. This is an indicator that they can move off-site as surface runoff or by leaching into the ground water. Where these herbicides are heavily applied on corn and other field crops, high concentrations in streams and rivers draining agricultural land may be detected after application. Of the over 600 pesticides currently in use, relatively few pesticides are commonly used for mosquito, black fly, and tick control. These pesticides include oils, biological pesticides, pyrethroids, carbamates, and organophosphates. Oil and biological pesticides such as Bacillus Thuringiensis (Bt) are widely used for mosquito control in the larval stages. The health impacts of oils are as varied as for those found in petroleum hydrocarbons. The more aromatic the fraction is - the more severe and negative the health impacts. There are many Material Safety Data Sheets that indicate the percentages of ethyl benzene. Pesticidal strains of the bacteria Bacillus Thuringiensis (Bt) is also used extensively to control black flies, mosquitoes, and gypsy moths in the larval stage. The varieties of Bt that are used as pesticides do not present a human health issue. However, it is recommended that Bt be linked to the CRP Ecological Technical Work Group report considering the ecological long term effects of pesticidal use. Commercial applicators use the carbamate carbaryl for the non-larval stage control of gypsy moths. Products containing carbaryl and the pyrethroid permethrin are widely applied for tick control in NJ.

Stressor-specific impacts considered including key impacts	Synthetic Pyrethroids such as d-phenothrin (sumithrin) have relatively low toxicity to humans. Permethrin is considered moderately toxic depending on the composition of the formulation. All of the pyrethroids can be absorbed through the skin, so direct contact should be avoided. Resmethrin, permethrin and phenothrin pyrethroids do not contain cyano groups that have been suspected of causing seizures or parasthetic effects. These synthetic pyrethroids are considered to be moderately persistent. Most pyrethroids are considered to be moderately to highly irritating to the eyes causing stinging, tearing, and blurred vision. At high doses, symptoms such as a stuffy runny nose, coughing, headache, fever, irregular heartbeat, nausea, weakness, and tremors may be experienced. Permethrin and bifenthrin are considered to be weak carcinogens. Synthetic pyrethroids have been the alternative to the banned pesticides, such as DDT, and exhibit similar toxicological mechanisms to those of DDT. However, they are far less persistent and more degradable. From high dose animal studies, adverse effects include reproductive, immune and neurological effects. Developmental effects such as neuroreceptor alteration leading to hypersensitivity, reduction in neural signal transmission efficiency and behavioral abnormalities may be seen in prenatal and neonatal nervous systems. Disturbances in the endocrine system, lymphocyte function, and thymus weight may contribute to immune response suppression. Future studies should include transgenerational effects of pyrethroids. Many organophosphates (OPs) and carbamates that are ranked as highly acutely toxic pesticides can be inhaled ingested or absorbed through the skin. Acute symptoms of OP poisoning develop within minutes to hours after exposure with symptoms first appearing in the respiratory system. Since the enzyme acetylcholinesterase is inhibited, the symptoms are related to effects on the central nervous system, muscarinic, and nicotinic receptors. Enzyme depression, in the case of plasma pseudo-cholinesterase, generally persists for several days to a few weeks; red blood cell enzyme activity may remain depressed for 1-3 months after over-exposure. Malathion, one of the OPs having a low acute mammalian toxicity, has low persistence especially in water. However there are several breakdown products with higher toxicity than the parent. Temephos is an organophosphate with low toxicity to mammals. It may bioaccumulate. It is of low persistence in water, low to moderate in soil, and high in plants. Diazinon, is an OP with low to moderate persistence. Use was diminished as of March 2001. Chlorpyrifos is an OP with moderate persistence and because of federal review and agreements, household use of chlorpyrifos will be drastically cut. Azinphos-methyl (brand-name is Guthion) and methyl-parathion has been the subject of federal agreements that will curtail agricultural use Acephate, is an acutely toxic OP that is under federal review. Phosmet, dimethoate, terbufos, some of the other OPs that are agriculturally are under review. Trichlorfon and chlorpyrifos are two OPs that account for about 6600 pounds of the golf course insecticide use. Fungicides account for 84% of the pesticide use on golf courses in NJ. The fungicide chlorothalonil accounts for over 98,000 pounds of the almost 237,000 pounds of total pesticide use on golf courses. DEP/PCP monitoring has detected in many of the pesticides used on the golf courses in golf course ponds and surface runoff. Many of the pesticides are not monitored routinely because of the difficulty and expense of their analysis. Vinclozolin, iprodione, DDVP, metalaxyl, and even DDT has been detected.

Exposure Assessment	Oils and petroleum hydrocarbons, are extensively used for agricultural, and horticultural uses and mosquito larval control. Solvents such as petroleum hydrocarbons are commonly used to aid in solvating the pesticides that are not very water soluble. They are considered to be "inert", yet their human health impact needs to be considered and would be similar to impacts of petroleum hydrocarbons. Methoprene is an insect growth regulator and is a long chain hydrocarbon ester. The toxic impact of hydrocarbons on human health is low, however, respiratory stress for sensitive subpopulations needs to be considered. The effects of synergists and inerts need evaluation. Synergists are substances added to pesticide formulations to increase the toxicity of the product. Synergists such as PBO and MGK 264 may be added to pyrethroids to increase the toxicity by interfering with their detoxification. Inerts are used as carriers, solvents, and stabilizers. Solvents in pyrethroid products include petroleum hydrocarbons, and aromatics such as Stoddard solvent and ethyl benzene.
Exposure routes and pathways considered (include indoor air as appropriate)	The exposure route is mainly by ingestion through drinking of water from wells and surface-water public-supply drainage basins that contain pesticide residues. Ingestion of bioaccumulated residues as a result of water contamination in e.g., food and fish, is not considered in this overview. The extensive and in-depth risk assessments for organophosphates are available on the EPA website and are continually being updated. Ultimately 40 OPs will be assessed. Most of these actions are being triggered by the 1996 FQPA regulation that specifically considers risk to children and infants. On the national scale, EPA started to formally re-review the risks-benefits of organophosphates. Chlorpyrifos, the highest homeowner use organophosphate termiticide and insecticide, has been determined to be unsafe for most homeowner use and especially for children. The manufacturer was urged to withdraw it from all home and garden uses. (EPA May, 2000). The registrant has not agreed to this as of December 2000. However, severe curtailment in use should decrease contamination. Another organophosphate of major use in NJ undergoing major review and assessment is diazinon. In December, 2000, EPA obtained an agreement that eliminates all indoor uses of diazinon effective March 2001, and starts a phase-out of lawn and garden uses yielding at least a 75% reduction in diazinon's use nationwide.
Population(s)/ecosystem(s) exposed statewide	NJ's residents drinking water from ground water wells and surface water contaminated with pesticides may be exposed. Those drinking water obtained from shallow ground water wells in areas with various pesticide uses may be at increased risk. There is an ecological link for the animals and aquatic life associated with impacted streams and rivers.
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	The NJDEP/Pesticide Control Program periodically and routinely performs surveys of the amount of pesticides applied by private applicators such as farmers and licensed professionals. The surveys do not include any pesticide use performed by private citizens. The survey found that a total of over 63 thousand pounds of pesticides were applied for mosquito control in NJ in 1998. This was before the West Nile-like Virus outbreak of 1999, that was accompanied by increased mosquito control. A new survey will be conducted to fill this data gap. The largest use of pesticide was oil

	(73%). Oil containing petroleum hydrocarbons could increase air volatiles and ground level ozone, and affect water bodies where the oil is spread as a larvicide. The next highest use pesticides are the organophosphates malathion (14%) and temephos (6%). While the last survey of 1998 did not indicate a high use of the synthetic pyrethroids, it is anticipated that their use will increase in future NJ mosquito control efforts. The results of mosquito pesticide use surveys for the years 1999 and 2000 was available in the Spring of 2001. Amounts of pesticides applied to lawns and turf by commercial applicators are tracked by the NJDEP/Pesticide Control Program. Over 518,000 pounds of pesticides were commercially applied in 1998. Of that amount used, 65% were herbicides. Phenoxy acid herbicides such as 2,4-D and mecoprop, and the dinitroaniline herbicide pendimethalin are widely used. About 42% of the insecticides used were represented by two OPs - chlorpyrifos and trichlorfon. A large data gap is the public's use of "over-the counter" pesticides bought in stores. The agricultural use of pesticides by over 2400 licensed private applicators was compiled by NJDEP/PCP. The total amount of pesticides in NJ used in 1997 was 1.4 million pounds. Sulfur 16% and the fumigant metam-sodium 221,700 pounds made up 15% of the state's total use. Many compounds, including fumigants, are not routinely monitored in NJ; therefore, there is no database available for evaluation. Pounds of Pesticides used on golf courses in 1996 was 236,844; agricultural usage accounted for over 1.4 million pounds (1,432,006 pounds). 52,409 pounds were used for rights of way treatments. Commercial lawncare and turf applications accounted for over 518,475 pounds of pesticides applied, and 63,436 pounds were used for termiticide control. Over 237,000 pounds of pesticides used on NJ golf courses was reported to the DEP/Pesticide Control Program. Of this amount, about 199,300 pounds or 87% were fungicides. Chlorothalonil contributes 85,700 or 36% and mancozeb another 26,500 or 11% to the total pesticide burden from pesticide use on golf courses. Chlorothalonil has been found in NJ surface water. Mancozeb is an EBDC type of pesticide and quickly degrades into the carcinogen ETU (ethylene thiourea) which is not monitored in NJ. Vinclozolin is another fungicide used (5700 pounds or 2.4% of total) and is being reviewed on the federal level on account of its negative impacts on the human endocrine system. Quintozene or pentachloronitrobenzene (5400 pounds or 2.3% of total) is a highly chlorinated compound that is not routinely monitored. A rough estimate can be made that 1% to 5% of the pesticide applied may move offsite associated with surface water runoff. Besides transport into ground water and surface water, volatilization also may occur. There are pesticide residues that will remain on crops and enter our food supply. (See pesticide residues and food write-up). Although pesticide use is being tracked, the monitoring system has not been expanded to keep up with the number of pesticides and the different sources. For surface water, so much of the detection of pesticides depends on the runoff from a specific time and site. For monitored pesticides that have MCLs or Health Advisory Levels or other target levels in the low parts-per-billion, pesticide levels in surface water have approached and occasionally exceeded these levels.
	Quantification of pesticide exposure levels is not currently possible and this identifies a serious data gap. Specific programs and research have not been established to address the data gap in the numbers of the general population

	expose of any identified vulnerable subgroup and the exposure levels. Also, data on specific areas or acreage, or numbers of people exposed in each county are not available. Degradation into pesticide products that are equal, more, or less toxic than the originally applied parent compounds is possible. With few exceptions, these pesticide degradation products are not routinely monitored so that assessment of these is not possible.
Specific population(s) at increased risk	The state's population in mostly rural areas derive their drinking water from shallow ground water wells. This risk is increased for those that drink ground water derived from vulnerable water wells. These are generally identified as sensitive shallow aquifers or outcrop areas that are in areas of increased pesticide use (as in southern NJ). Infants, children, and the elderly may be at increased risk. Sensitive individuals such as those with chronic lung disease, (e.g., asthma), CNS dysfunction, sensitivity to specific pesticides or formulations could experience adverse effects or an increase in their symptoms. There may be increased effects for people with sensitive or compromised immune functions. There may be a socioeconomic link between the risk from drinking polluted water and economic ability to mitigate it For example, many of these shallow vulnerable wells are in very rural and agricultural areas and the residents are not able to afford drilling deeper wells that tap into cleaner aquifers or installing water filters.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	More research is needed to address the data gaps that make it impossible to quantify the human health impact of that exposure for the identified vulnerable subgroups in NJ. The actual levels of exposure to pesticide would be similar to that of exposure levels found for each source and pesticide in the general population. However, the assessment of the impacts of the mixtures of pesticides and related products presents a huge challenge.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	
Risk Characterization Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	USGS (May 1999) reported that every water sample collected from 50 NJ and LI, NY streams in June, 1997, indicated the presence of at least one pesticide. Of the 47 pesticides that were monitored, at least 25 were found. 93% of the samples contained atrazine, metolachlor (86%), prometon (84%), simazine and desethyl-atrazine (a chloro-metabolite of atrazine) - (78%), and over 40% indicated the acutely toxic OP diazinon and the carbamate carbaryl. These pesticides were found in the agricultural areas. Pesticides associated with turf and lawncare such as the nitroaniline type chemicals, benfluralin and trifluralin.
	The median concentration of 0.2 ppb (micrograms per liter) for the 25 pesticides found in surface water (USGS, 1997) was related to the amount of agricultural land draining into a water basin.

	In an earlier smaller study (USGS, 1996) relating public water supplies and wells to pesticide contamination, 4% of the almost 2000 ground water-based public community supplies in NJ were considered vulnerable to pesticide contamination. Five out of 23 pesticides were detected in the public water supply wells. The chloracetanilide pesticides metolachlor and metalaxyl, the triazines atrazine and simazine, and the dinitrophenolic compound dinoseb were detected. Dinoseb is a highly toxic herbicide and insecticide that was cancelled in1986. The adverse effects included high acute toxicity, birth defects, and male sterility. A federal MCL or HAL is not available. Eighty percent of the surface water public community supplies are considered vulnerable. Ten pesticides were detected out of the 23 pesticides that were analyzed. The pesticides most frequently detected were the chloracetanilides alachlor, metolachlor, and the triazines atrazine and simazine. Pesticides have not been found in drinking water from surface sources under the regular testing required by public drinking water purveyors. In public drinking water from groundwater sources pesticides are rarely seen, and have not been detected above the MCL.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate) Size of population(s) affected	Unknown - Moderate Effects of pesticide residues in NJ waters is difficult to evaluate because of inadequate or non-existent information of the biological and health effects of low levels of mixtures of pesticides, pesticide transformation products, and seasonal exposures. Assessment is also hampered by lack of pesticide monitoring and trend data on water concentration levels of pesticide degradation products and the resulting exposures. EPA has now adopted a policy where in the absence of contrary information, the degradation products will be considered at least as toxic as it's parent pesticide. Another new concern in the initial stages of being addressed is the multigenerational pesticide effects and endocrine disruption effects of pesticides. EPA has concerns about long-term health effects of the chlorinated cyclodienes including effects on the liver and the Central Nervous System (CNS), and causing an increase in the risk of cancer. Persistent pesticides are still being found many years after their use stopped and may drift global distances from the original application points (e.g., Canada, arctic). NJDEP/PCP monitoring indicates pesticides associated with use in the past are still entering surface waters; DDT and its degradation products DDD and DDE were found in surface water at a golf course site. Dieldrin does not have MCLs or HAL to exceed however, it is still being detected at levels in surface water (USGS, 1997) that are above the state and federal levels set for aquatic life protection or human health criteria. High Atrazine and metolachlor is among one of the most commonly detected pesticides in NJ and USGS monitoring studies. In a comparative land-use study, these 2 agricultural pesticides were found in over 90% of samples taken from streams and in about 40% of ground water samples taken from agricultural areas. Pesticides associated with both agricultural and urban use such as simazine, was detected in both ground and surface water from both agricultural and urban areas. Prometon was found frequently in urban shallow ground water and in about 60% in urban and agricultural stream samples.
	The pesticide atrazine is classified as a possible human carcinogen. During most of the year, atrazine concentration in ground water and surface water is usually less than the drinking water maximum contaminant level of 3 ug/L (3 ppb) set for public drinking water supplies. However, in spring and early summer this level may be exceeded when

	applications to agricultural land are lost as runoff to surface waters. Further consideration is warranted when the commonly detected chlorinated degradation products of atrazine are included into the estimate of exposure levels for atrazine. The toxicity of the several chlorinated degradation products is considered to be equivalent to atrazine and in some states (MN) they are included as part of the reported concentration levels that are subject to regulation. The levels from limited ground water monitoring studies indicate that the degradation products may be found in even higher levels than atrazine itself. Currently, the atrazine and other triazines are undergoing major federal review. It would be difficult to accurately estimate the size of population affected because many rural farming communities rely on well water as their drinking water source, whereas urban communities rely on potable water from pubic-water-supply reservoirs.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	High The public uncertainties in the assessments are high mainly because of the wide spread data gaps for both the exposure data of susceptible subgroups to many pesticides in various indoor environments and the lack of data in the assessments of resultant and cumulative risks for so many pesticides. There is insufficient data and knowledge of the impacts of pesticides; what concentrations of which pesticides and formulation solvents and "inert ingredients" are necessary to predict the pesticide impacts. Much of the pesticide impact data is based on data obtained from toxicological testing utilizing high doses of the pesticide in animals. EPA, among others, has serious concerns about the chronic impacts of low doses on especially the endocrine system and reproduction, the neurological and immune systems, cognitive and behavioral systems such as learning, and memory.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	High There are changes in risk estimates that are based on recent re-assessments. Additional data may be expected to uncover additional subpopulations at risk. Full risk re-assessments are being carried out by EPA for the organophosphate and triazine pesticides. Efforts at assessing endocrine disruption by pesticides will also uncover new risks. Multiyear assessments examine population exposure levels from all media and sources and both human health and ecological considerations. Future assessments should take into consideration the decrease in risk caused by withdrawal or cancellation of high risk pesticides, and the new risks posed by the use of substitutes to control pests.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	+++ The potential for future change in the underlying risk from atrazine is dependent on changes in the regulation controlling the use of this pesticide, and changes in the practice of farmers and other users. Regulatory changes may involve further revision of label which dictates the legal use. On the other hand, farmers can reduce runoff losses by adopting best management practices that include IPM, setting buffer strips to trap runoff, conservation tillage to reduce soil erosion, reducing application rate, timing of applications so as not to be followed immediately by rain, subsurface drainage, contour planting, crop rotation, and stripcropping.
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	Low -The likelihood of any catastrophic event with adverse effect on humans is low. A severe impact on water could result from a spill, storage or transportation accident. Although some pesticides in surface waters are rarely detected, there are others that may exceed the maximum contaminant level (MCL) during the spring and early summer when applications are made to agricultural land. However, their annual average concentrations seldom exceed the regulatory standard established by EPA.
Extent to which risks are currently reduced through in-	Low - Pesticide use is currently being regulated under the federal regulation (FIFRA) and state regulation under the NJDEP Pesticide Control Program. However, because much of the contamination is being created as a result of legally

place regulations and controls	allowable pesticide use, it's clear that new regulations and Pesticide Management Programs need to be instituted regarding pesticides with the potential to contaminate ground water and surface water.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	M - lawn and turf care; pesticide mixing/loading areas; distributorships; pesticides applicator businesses; golf courses; also through spills, leaks, and improper disposal.
Small business industry	M - lawn and turf care pesticide mixing /loading areas; distributorships; pesticides applicator businesses; golf courses; also through spills, leaks, and improper disposal.
Transportation	M -rights of way pesticide applications for weed control or removal.
Residential	High - pesticide applications by homeowners to residential property; improper use and disposal.
Agriculture	High - pesticide applications for agricultural, horticultural, silvicultural or forestry, farms, improper use and disposal
Recreation	M - pesticide use in public play areas and parks for pest and disease vector control.
Resource extraction	
Government	M -Pesticide use for disease vector control; NJ County mosquito control; increasing pesticide use to control West-Nile Virus.
Natural sources	
Contaminated sites	M -landfills and disposal sites contribute to pesticide; especially for older banned or cancelled pesticides.
Diffuse and non-NJ sources	
Sediment	
Soil	
Non-local air sources (including deposition)	Pesticides are being found transported both short and long distances by evaporation into the air by fog, mist, and rain.
Biota sinks	

Issue: Pesticides-Water
Author: Areta Wowk
Version: 05/01
Human Health Issue Summary: Pesticides - Water

What is it?
Pesticides are used in agriculture to increase yield and decrease costs. Unfortunately, pesticide residues can contaminate drinking water supplies. The wide range of pesticides in use makes it difficult to pinpoint individual health endpoints. However, chronic pesticide health effects include nervous system damage, immune, visual, respiratory and cancer effects.

Who's at risk?
A USGS survey found that every drinking water supply from sampled NJ, LI and NY streams contained at least one pesticide. In a different study, 4% of New Jersey wells were considered vulnerable to pesticide contamination. In these studies, several different pesticides were identified. There are several health endpoints from the different contaminating pesticides. However, levels of pesticides in New Jersey drinking water do not exceed and rarely, if ever, approach regulatory levels.

What is the extent of human health problem in New Jersey?
Regular testing of drinking water from public drinking water suppliers has not identified contamination above standards established to protect public health. However, a lack of information on the effects of low level mixtures, transformation products and seasonal exposures suggests the possibility for greater risk.

What's being done?
Pesticide application is regulated through a federal licensing process. Drinking water is monitored to identify samples of elevated contamination. Further research is needed to better understand the possible impacts from low level contamination.

Risk Summary
Almost the entire state population is exposed to possible pesticide contamination, but the level of contamination is low and the probability of health effects is not certain. These factors led the Technical Working Group to assign a Medium ranking of health risks.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
3	5	5	4

Issue: Pfiesteria
Author: Tom Atherholt
Version: 12/19/00

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Assessment

Risk Assessment Framework	Findings
Hazard Identification	**Pfiesteria**
Stressor	*Pfiesteria piscicida*. An estuarine unicellular microorganism that can, under certain conditions, multiply and produce toxins which can cause lesions and death in fish and can cause adverse human health effects to exposed individuals. This brief review focuses on the adverse human health effects (Burkholder and Glasgow, 1997; Burkholder, 1999). All ecological effects are covered in a separate *Pfiesteria* document authored by Mary Gastrich for the Ecological Quality Technical Work Group.
Description of stressor (including etiology)	*Pfiesteria piscicida, Pfiesteria shumwayae* and several other, as yet unidentified toxin-producing species are a group of estuarine unicellular dinoflagellate microorganisms. Dinoflagellates are common components of plankton although *Pfiesteria* are not normal components of plankton. Some *Pfiesteria* species are not capable of producing toxins. Other *Pfiesteria* are not normally toxic but are able to produce toxins in the presence of large numbers of fish and favorable environmental conditions such as warm, calm waters, low dissolved oxygen and possibly high concentrations of phosphates and nitrates.

Stressor-specific impacts considered including key impacts	Definitely one and perhaps 2 or 3 separate toxins are produced, although these toxins have not yet been characterized or conclusively linked to *Pfiesteria*. The toxins do not appear to last long in the environment nor do they accumulate in fish tissues or organs. Adverse human heath effects were first observed in laboratory personnel working with *Pfiesteria* cultures. There have also been numerous Anecdotal reports of adverse effects in fisherman exposed during fish kill events. The various adverse health effects that have affected all exposed laboratory workers (and some of the fisherman) include memory problems, emotional changes, skin lesions and asthenia (loss of strength). Other adverse effects that have effected some but not all of the exposed workers (as well as some of the fisherman) include; tingling and/or burning sensation in the extremities and around the mouth, headache, nausea, abdominal pain, tearing/eye irritation, respiratory problems, joint pain, muscle pain, vomiting, and perspiration. Unfortunately, there is no consistent suite of symptoms unique to *Pfiesteria* (Glasgow *et al.,* 1995). Affected workers have complained of disorientation with a sense of depersonalization and of being compelled to continue and lacking an ability to recognize that something was wrong. All have short-term memory loss lasting from 1 to 8 weeks. Heavily exposed lab workers have had residual symptoms, following vigorous exercises, that have lasted for years afterward (Glasgow *et al*, 1995).
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	There is no evidence that eating fish or shellfish from a fish kill area causes illness but it is not advisable to eat fish with epidermal sores from fish kill areas. Exposure to the toxins occurs by direct contact of the skin to the toxin-containing waters during a *Pfiesteria*-related fish kill event and/or by inhaling aerosols (as from an open boat) in the immediate fish kill area.
Population(s)/ecosystem(s) exposed statewide	There have not been any documented *Pfiesteria*-related fish kill events in NJ as of December, 2000 and no documented cases of adversely affected individuals (Atherholt and Ruppel, 2000; NJDHSS/NJDEP, 2000). The potential exposure group, given the isolated and temporal nature of the fish kills, would be expected to be small - anywhere from 1 to 10's of persons.
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	See ecological assessment for *Pfiesteria* concentrations in laboratory studies and several documented fish kill events.
Specific population(s) at increased risk	There are no known human subpopulations at increased risk although such populations may exist.

Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	Same as for general population.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	No data are available to derive a dose-response relationship.
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	Given the isolated and infrequent occurrence of *Pfiesteria*-related fish kill events, including the fact that there have been no documented *Pfiesteria*-related fish kills in NJ to date, the risk to potentially exposed individuals is considered to be very low. Aside from a few affected laboratory workers, only a handful of fisherman, approximately 25, have reported adverse health effects following exposure in *Pfiesteria*-related fish kill areas (Glasgow *et al*, 1995).
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Most of the above enumerated symptoms are A significant although perhaps not severe. The symptoms are reversible although there appears to be residual effects in heavily exposed laboratory workers that can last for years following exposure (see above).
Size of population(s) affected	Small. Probably less than 50 nationwide and, to date, none in NJ.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	Low.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	Low.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	0
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	None.
Extent to which risks are currently reduced through in-place regulations and controls	Risk of a *Pfiesteria* related event in NJ is considered low primarily because there are few estuary locations in NJ with the right set of environmental conditions - conducive to *Pfiesteria* multiplication. In addition, secondary wastewater treatment along with treatment plant outfall (effluent) pipes which extend far offshore along the entire

	NJ coast, coupled with a low number of concentrated animal feedlot operations, help to keep the potential for nutrient overloading in check. Nevertheless, in the unlikely event of a *Pfiesteria*-related fish kill in NJ, the Departments of Health and Senior Services and Environmental Protection have assembled a contingency plan (NJDHSS/NJDEP, 2000). The plan provides background information on *Pfiesteria* and specifies the protocol to be followed by NJDHSS, NJDEP, the Coast Guard, NJ Marine Police, and county and local health and emergency response personnel in the event of a *Pfiesteria* fish kill.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	L (possible nutrient loading from wastewater treatment plant effluents)
Small business industry	L (possible nutrient loading from wastewater treatment plant effluents)
Transportation	none
Residential	L (possible nutrient loading from septic leachates, lawn fertilizer runoff)
Agriculture	L (possible nutrient loading from animal waste run-off)
Recreation	none
Resource extraction	none
Government	none
Natural sources	L. *Pfiesteria* are natural components of the estuarine environment.
Contaminated sites	none
Diffuse and non-NJ sources	L. (storm water runoff)
Sediment	L. Possibly present in some estuarine sediments.
Soil	none

Non-local air sources (including deposition)	none
Biota sinks	none

Issue: Pfiesteria
Author: Tom Atherholt
Version: 12/19/00

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
L or 1	L or 1	None known, but may exist	L or 1
			L or 1

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Issue: Polychlorinated biphenyls (PCBs)
Author: Perry Cohn, Gloria Post
Version: 06/04/02

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Polychlorinated biphenyls (PCBs)**

Description of stressor (including etiology)	PCBs are a group of 209 polychlorinated biphenyl congeners produced as mixtures with varying degrees of chlorination for different uses, such as fire retardants, hydraulic fluids, insulating materials in transformers and capacitors, and open-end applications (e.g., plasticizers, surface coatings, inks, adhesives, pesticide extenders, paints, and microencapsulation of dyes for carbonless duplicating paper). About 130 of these were produced in commercial products. They are no longer being produced as of the end of 1978. By1976, only 5% of the transformers produced in the U.S. were filled with PCBs; however, 95% of the capacitors produced in the U.S. were filled with PCBs (IARC, 1978, cited in ATSDR, 1997). Nevertheless, the life expectancy of transformers containing PCBs is greater than 30 years, and the life expectancy of capacitors ranges from 10 to 20 years, depending on the electrical application. In 1981, an estimated 131,200 transformers containing PCBs were in service in the U.S., representing approximately 1% of all operational transformers. Small amounts of PCDDs and PCDFs were by-products. PCDDs and PCDFs can also be formed by heating PCBs, such as occurs during a transformer fire. Certain congeners are found in the environment, especially in fish and animals that consume fish, including humans. Since congeners with less chlorination are more easily metabolized and excreted, congeners remaining in the body, particularly in adipose tissue, have a greater degree of chlorination. In humans, PCBs typically co-occur with PCDDs, PCDFs, PBBs, PBDEs, PCNs and various other organochlorine pesticides. The levels of PCDDs, PCDFs, and PBBs are lower in the general population than occupational cohorts and are harder to measure.
Stressor-specific impacts considered including key impacts	Among the effects of the various of the PCB congeners are neurodevelopmental retardation, decreased thyroxine levels, reproductive dysfunction, immune system suppression, carcinogenesis, and enzyme induction. Some of these effects are related to ability of congeners (mostly coplanar) to bind to the aromatic hydrocarbon receptor protein (AhR) that also binds dioxin. These effects are shared with other dioxin-like organochlorines. (The overall dioxin-like activity has been quantified by the assignment of toxic equivalence factor, a relative potency scheme assuming dose additivity, to each congener. Multiplying it by the congener concentration and summing all the congeners yields a toxic equivalent (TEQ) for a mixture.) There are several different classes of PCBs and their hydroxylated metabolites, each inducing different sets of enzymes and binding either to estrogen or androgen receptors to cause estrogenic, androgenic, or antagonistic effects. Similarly, many ortho-chlorinated (non-coplanar) PCBs and certain other organochlorines compete with thyroid hormones for binding to thyroid hormone receptors found in various organs, including the brain. A number of growth factors and their receptors are also affected by PCBs and organochlorines.
	The likely effects of PCBs (not entirely separable from co-occurring organochlorine compounds because of their low detectability in serum) include breast cancer, non-Hodgkin's lymphomas, liver and gall bladder cancers, pancreatic cancer, decreased circulating thyroid hormone, and prenatal effects that influence postnatal neurodevelopment. The fact that PCBs and various other co-occurring organochlorines have similar effects suggests the usefulness of a generalized risk assessment of organochlorines. <u>Cancers</u> Animals studies strongly indicate an increased risk of liver and thyroid cancer with exposure to an Aroclor 1254 mixture characterized by a large proportion of highly chlorinated PCBs. The cancer potency slope is 2.2 per mg/kg/day and 10^{-6} risk corresponds to $5x10^{-7}$

	mg/kg/day. Mixtures with less chlorination had lower potency. Central potency slope estimates of combined liver adenomas and carcinomas in female rats with the linearized multi-stage model were 0.04, 0.3, 1.2, 0.4 and 1.6 per mg/kg/day and 95% upper bound slope estimates were 0.07, 0.4, 1.5, 0.5, and 2.2 per mg/kg/day for Aroclors 1016, 1242, 1254, 1260 (Cogliano, 1998), and 1260 (Moore et al., 1994), respectively. Male rats only responded to Aroclor 1260, but thyroid follicular adenomas and carcinomas were increased after expose to Aroclors 1242 and 1254 in a dose-response trend in male rats only. Arochlor 1254 and 1260 are made up of approximately 50% and 10% penta-chlorinated PCBs, 25% and 40% hexa-chlorinated PCBs, 5% and 40% hepta-chlorinated PCBs, and none and 5% octa-chlorinated PCBs. Individual PCBs or mixtures with higher chlorination have not been tested. Of the internal cancers, liver, gall bladder and biliary tract, rectal and kidney cancers have been acknowledged as those most frequently found at higher than expected levels in the combined results of eight occupational mortality studies (Longnecker et al., 1997). Liver and biliary cancer had a collective SMR=1.6 (95%CI, 0.8-2.7) and kidney had an SMR=1.8 (95%CI, 1.0-3.0). Rectal and pancreatic cancers had SMRs of about 1.5. In the YuCheng poisoning cohort in Taiwan, liver cancer was increased after five years of follow-up. Case-control epidemiology has observed a link between PCBs (actually, organochlorine concentration as measured by PCBs) and breast cancer (among the 10-15% of women with a CYP1A1 polymorphism), non-Hodgkin's lymphoma (especially among those with elevated seropositivity to Epstein-Barr virus), and pancreatic cancer (especially among those with a mutation in the k-ras gene). These are described in more detail in the Appendix. <u>Neurodevelopment</u> There is evidence that prenatal and postnatal exposure to PCB mixtures and congeners in laboratory animals, as well as organochlorines measured in humans as PCBs, have detrimental effects on neurodevelopment (reviewed by Schantz, 1996). These are described in more detail in the Appendix.
Exposure Assessment	
exposure routes and pathways considered **(include indoor air as appropriate)**	PCB/PCDD/PCDF contaminated fish, breast feeding.
population(s)/ecosystem(s) exposed statewide	sport fish consumers, but also general population through dietary exposure and infant breast feeding
quantification of exposure levels statewide, including populations at significantly increased exposure **(include indoor air as separate category as appropriate)**	There is no information about blood or adipose PCB levels in New Jersey, but they are probably similar to recent studies in other states. Mean PCB levels in the 1990s studies around the Great Lakes were 5 and 14 μg/L (roughly 1.5 and 5 μg/g lipid) among non-consumers and fish eaters, respectively (Anderson et al., 1998; Humphrey et al., 2000). This would correlate with back-calculated daily ingestion, assuming 10 year half-life and first order kinetics (as described in detail below in quantitative dose/impact-assessment employed), of 0.07 and 0.24 □g/kg/day, respectively. Maximums were about 30 and 70 μg/L, respectively. In the western New York breast cancer case-control study, levels among controls were 0.7-19 μg/L (0.1-2.6 μg/g lipid, median = 0.6) or 0.026 μkg/day at the

	median (Moysich et al., 1999b). In the Maryland case-control study on NHL median serum level was 815 µg/kg lipid or 0.039 µg/kg/day. In the San Francisco Bay area case-control study on pancreatic cancer the upper tertile cutpoint serum level was 360 ng/g or 0.017 µg/kg/day. Levels of organochlorines and chlorine content increase with age and decrease with the number and duration of lactation periods. The FDA Total Diet Study, which assumes that the average adult male weighs 70 kg and that the estimated dietary intake of total PCBs is 0.0005 □g/kg/day (1982B1984 data), the average daily dietary exposure would be 0.035□g. According to Fensterheim (1993), PCB intake from food was 0.0007 □g/kg/day for the years 1987B1989. This would make the average daily dietary exposure 0.05□g. Meat products are the major contributing source, 85% of total dietary intake (Gunderson 1988), but fish consumed by recreational and subsistence fishers are not included in these studies. In the newest survey beef and pork contain about 0.02 □g/kg (ppm), while canned tuna contains an average of 0.045 □g/kg (FDA, 2000). Leaner meat would contain less. Breast feeding transfers organochlorines from mother to infant (as much as 20-25% of prenatal maternal body burden) and results in organochlorine intake in the range of 50-fold higher than adults on a body weight basis. Most of the ingested organochlorine is absorbed.
specific population(s) at increased risk	<u>Cancer</u> Breast: women with P4501A1 (CYP1A1) polymorphism (valine->isoleucine substitution in exon 7), affecting 10-15% of the population. NHL: people seropositive for Epstein-Barr virus Pancreatic: people with mutated k-ras gene. <u>Neurodevelopment</u> Pregnant women and their fetuses.
quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations)	Same as above for statewide levels. There are fish consumption advisories for pregnant women that may modify the diet.
Dose/Impact-Response Assessment	
quantitative dose/impact-assessment employed for each population considered	Back-calculated daily ingestion dose (D), based on serum, adipose, or breast milk levels D at the cut-point used in analysis, is used as a basis for a NOAEL or as a dietary benchmark for cancer along with the population attributable risk (PAR). Assuming that dietary PCBs are retained in adipose tissue with a half-life of 10 yr, that adipose tissue is 25% of body weight, and first order kinetics for PCB turnover, and by equating the concentration in blood or breast milk, as □g/kg lipid, to the concentration, □g/kg lipid, in adipose tissue, a daily intake of PCBs can be back-calculated. The equation for back-calculation, based on a simplified toxicokinetic model (Tilson et al., 1990; Great Lakes Sport Fish Advisory Task Force, 1993) is: D = (□g/kg lipid) x 0.25 x ln 2(= 0.693)/365 days x 10 yrs

	The equation for PAR is: PAR = P_E x (OR or RR - 1)/(P_E x (OR or RR-1) + 1) (P_E is the joint probability of exposure and susceptibility. For example if the affected group are those above the median serum PCB level and the 10% in a susceptible subpopulation, P_E would be 0.5 x 0.1 = 0.05.) The annual attributable incidence rate = PAR x annual rate.
Risk Characterization risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	The co-occurrence of the various organohalogens, the difficulty of measuring all of them, and the resulting lack of complete body burden data means that PCB congeners, as the contaminants with by far the highest concentrations, are best viewed as surrogates. This may not be a problem in assessing risk, however. For example, although the various dioxin-like PCB congeners are significantly less toxic than 2,3,7,8-TCDD (by TEFs), their overall toxic activity (TEQ) is greater due to higher concentration. In addition, fish, as the major source of dietary PCBs, have a similar mix of organochlorines. In comparing the following population-based studies to the rat studies, it should be noted that the proportions of congeners grouped by degree of chlorination in studies in the Great Lakes approximate that in Aroclor 1260 (Anderson et al., 1998; Humphrey et al., 2000). Those proportions were similar in both fish-eaters and controls. **Breast cancer**: Among women with the P4501A1 polymorphism, there was an association (adjusted OR=2.9) between the incidence of breast cancer and PCB levels >3.7 ng/g, above the median (Moysich et al., 1999). The population attributable risk (PAR) is 0.087 (0.1x0.5x(2.9-1)/(1+numerator)), based on 10% of women having the polymorphism and half (by definition) above the median. Based on a New Jersey population of one million women over age 50 (an approximation of postmenopausal status), the Ajoint@ risk group is 50,000 (0.1 x 0.5 x 10^6), and, based on a PAR of 0.087 and an approximate rate of 3000 new cases each year among women over 50 in New Jersey, about 260 cases would be attributable to the joint effects of the polymorphism and serum PCBs >3.7 ng/g. Assuming that premenopausal women had the same risk, then, based on roughly 6,000 new cases per year in New Jersey, roughly 500 cases would be attributable to the joint effects. At the median blood concentration, back-calculated daily intake is $2.6x10^{-5}$ mg/kg/day.
risk estimate(s) by population at risk, cont.	**Non-Hodgkin's lymphomas**: A case-control study has linked non-Hodgkin's lymphomas to PCBs (Rothman et al., 1997) with an OR, adjusted by DDT levels, socioeconomic factors and cigarette use, of 2.9 (95% CI, 1.1-8.1) and 4.3 (95%CI, 1.6-12) for the third and fourth quartiles (p_{trend} = 0.003). There was a positive interaction between seropositivity for Epstein-Barr virus and PCB concentration. Among those with both exposures, there was an OR of 22 (95%CI, 4-115). In the overall study the PAR is 0.75 ((0.25x(4.3 - 1)+0.25x(2.9 -1)) /(1+numerator)). Based on 1600 cases per year, 1200 were attributable to PCBs. The back-calculated body burden at the median (815 ng/g lipid PCBs) is $4x10^{-5}$ mg/kg/day. The joint effects correspond to an attributable risk of about 0.5, 800 cases. In the study of hairy cell leukemia by Nordström et al., 2000, the joint effect PAR is 0.5 (0.19x0.5x(11 - 1)/(1+numerator)) and the attributable annual risk is $2x10^4$ (800 cases/$4x10^6$). The back-calculated daily intake is $1.35x10^{-5}$ mg/kg/day.

	A small case-control study (Hardell et al., 1996) found that PCB congeners with higher degree of chlorination (PCB #153 and over) were present at higher concentrations in adipose tissue of patients with non-Hodgkin=s lymphoma (greater than the median1300 ng/g lipid: OR=2.7, 95%CI, 0.8-9.4). The PAR would be about 0.45 (0.5x(2.7 - 1)/(1+numerator)), or about 720 cases annually in NJ. Back-calculated daily intake is $6.1x10^{-5}$ mg/kg/day.
risk estimate(s) by population at risk, cont.	Pancreatic cancer: Pancreatic cancer was associated with total serum PCBs in a case-control study (Hoppin et al., 2000) with an OR in the highest tertile (≥360 ng/g lipid) of 4.2 (95%CI 1.8-9.4) compared with the lowest tertile. This corresponds to a PAR of 0.55 (0.3x(4.2 - 1)/(1+numerator)), and based on about 1700 cases per year in New Jersey, as many as 950 cases are attributable. Back-calculated daily intake is $1.7x10^{-5}$ mg/kg/day. In the highest tertiles (above approximately 0.25 g/g for each of the three predominant congeners), the risk of pancreatic cancer with a K-ras mutation is strongly linked to serum PCBs (Porta et al., 1999), with adjusted OR for occurrence of the mutated K-ras (versus wild-type) of 6.6 for PCB #138, 6.0 for #153, and 9.6 for #180. There were no significant differences between cases with wild type K-ras and controls. With no assumptions about those causal links, the PARs for the three analyzed PCBs among cases with the K-ras mutation in the highest tertiles were in a range of 0.4-0.55 (0.77x0.17x(range of ORs= 6.0 to 9.6 -1))/(1+numerator)), which would be about 700-900 cases. Since these congeners make up 2/3 of the total PCBs in this study, the back-calculated daily intake of total PCBs is $5.3x10^{-5}$ mg/kg/day. Summary: Summing over all the various cancers in these reviewed studies, about 2,000 to 2,500 cases of cancer per year are attributable to PCBs.
risk estimate(s) by population at risk, cont.	Neurodevelopment: Children born to women with PCB levels in the highest quartile (or higher percentile in North Carolina) had a statistically significant decrement in various neurological and mental ability assays. Although, most of the affected assays were not consistent across all studies, there was sufficient evidence to indicate that decrements do exist. Several possible NOAELs in the Michigan cohort were 2.5- $5.8x10^{-5}$ mg/kg/day. For neonatal hypotonicity of reflexes and poorer psychomotor performance at 12 months of age in the North Carolina cohort, the estimated NOAEL was $1.7x10^{-4}$ mg/kg/day. It is harder to determine a similar level for the Dutch study of neonates due to the different way in which analysis was conducted and because serum samples were not lipid adjusted. The OR for Aneuro-optimality@ and hypotonia among breast-feeding infants fed with breast milk containing >540 ng/g milk fat (approximately the top quartile) vs. breast milk with <540 ng/g was 3.4 (95%CI, 1.7-7.1). That would correspond to a NOAEL of $2.6x10^{-5}$ mg/kg/day. Significant differences in the Kaufman Assessment at 42 months corresponds to a NOAEL of $4.6x10^{-5}$ mg/kg/day. Differences on the Bailey Mental Development Index conducted at 7 months old in the German study occurred above and below median breast milk levels, corresponding to a NOAEL of $2.1x10^{-5}$ mg/kg/day. Usually a safety factor of 3-10 is used to account for inter-individual differences when using epidemiological evidence as the basis for determining a reference dose (RfD). The RfD would be in the range of 2-$17x10^{-6}$ mg/kg/day. However, if the McKinney method (2 PCB congener peaks) used for North Carolina yields values that are twice as high as Webb-McCall method (6 PCB congener peaks) used by the Michigan group, and if the Webb-McCall method is "truer", then the North Carolina estimate of the RfD should be cut by half (Schantz, 1996). The resulting range would be 2-$8x10^{-6}$ mg/kg/day. For laboratory animal experiments an additional safety factor of 10 is added to account for interspecies differences. Thus, an RfD based on neurodevelopmental deficits in the rhesus would be $1.4x10^{-5}$ mg/kg/day, which is about an order of magnitude higher than the human-based RfD. However, it would be consistent with the greater sensitivity of humans or the neurologic endpoints measured in humans to other neurotoxins, like lead and mercury.

assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	PCBs range from moderately persistent to highly persistent (highly chlorinated congeners), concentrating in adipose tissue. Breast-feeding of infants mobilizes adipose tissue organochlorines into breast milk. There is moderate evidence that fetal and postnatal PCB exposure causes long-term neurodevelopmental effects in children born to women in the top quartile of PCB body burden. The extent and persistence of these effects is still the subject of research. One long-term studies found persistence of neurodevelopmental decrements, while the other did not. About 1/3-1/2 of the incidence of breast, pancreatic and non-Hodgkin=s lymphatic malignancies are attributable to organochlorines as measured by PCBs. Liver and gall bladder cancers, elevated in PCB treated rats, occupational cohorts and the poisoning cohorts, were not studied in the general population.
size of population(s) affected	Breast cancer: About 10-15% of women carry a P4501A1 polymorphism that can synergize with PCB contamination in postmenopausal women (approximately a million over 50 in New Jersey) and the carriers would be about one hundred to one hundred fifty thousand women. About 1/3-1/2 of those would have PCBs high enough to cause a significant increase in breast cancer. Similar risks may also be present in premenopausal women.
	Non-Hodgkin's lymphoma: Persons in the upper half of PCB burden in the general population were associated with 1/3-1/2 of incident cases. In the 10% of the general population seropositive for EB virus, there is strong synergism with PCB burden. Pancreatic cancer: Persons in the upper half of PCB burden in the general population were associated with half of incident cases. There appears to be additional synergism with mutated K-ras oncogenes.
assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	Cancer: M Animals studies strongly indicate an increased risk of cancer with exposure to a mixture characterized by a large proportion of highly chlorinated PCBs. However, the PCB mixtures were probably contaminated with PCDFs. Most occupational studies indicate a significantly or marginally increased risk of skin, liver, gall bladder, rectal, kidney and pancreatic cancers with workplace exposure to commercial mixtures containing 41-54% chlorine but body burden was not measured. Case-control studies of the general population provide evidence of moderate uncertainty that risk of breast cancer and non-Hodgkin=s lymphoma are increased with PCB body burden, particularly among those in certain subgroups. While the body burdens of DDT/DDE and other organochlorines have been studied, PCDDs and PCDFs have not been as well characterized, since they occur at levels that are hard to measure in blood. PCDDs and PCDFs were measured in a study of non-Hodgkin=s lymphoma and adipose tissue PCBs and were not found to be associated. There is the possibility that exposures to more metabolizable, less chlorinated PCBs earlier in life may be causally associated, but due to the higher metabolism and turnover of less chlorinated PCBs, congeners with greater degrees of chlorination appear to be more associated.
assessment of uncertainties in this	Neurodevelopment: M

assessment (H,M,L) and brief description, and data gaps	Animals studies strongly indicate a negative effect on neurodevelopment following fetal exposure to individual PCBs and mixtures of PCBs. Human studies have looked primarily at maternal serum and breast milk levels, where persistent PCBs dominate, and may obscure the prior exposures to PCB mixtures containing PCBs with lower degree of chlorination. The effect of PCDDs and PCDFs have not been as well studied, except in a study of Dutch neonates where the analysis was based on combined dioxin and dioxin-like TEQ (toxic equivalent = toxic equivalent factor for each congener x congener concentration, based on measurements of various dioxin-like effects).
potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	H: More population-based studies need to be done to confirm the current studies. Identification of susceptible subpopulations in the case-control and occupational cohorts will help elucidate the real risks. Better characterization of body burden of PCDDs and PCDFs will help.
potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , ≈, where + is improvement)	+, organochlorine body burdens are decreasing
potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L(?), only due to fires/explosions of capacitors and transformers still containing PCBs. The estimated number remaining is probably tiny. PCDDs, PCDFs, and PCNs are produced by incineration of certain plastics.
extent to which risks are currently reduced through in-place regulations and controls	fish consumption advisories, FDA limits in foods.
Relative Contributions of Sources to Risk (H,M,L)	fish: H; other foods: M
Allocation of stressor-specific risk to primary NJ sources	
large business/industry	M
small business industry	M (old transformers & capacitors, electrical contractors)
transportation	L
residential	L
agriculture	L

recreation	L
resource extraction	L
government	L
natural sources	L
contaminated sites	M
diffuse and non-NJ sources	M
sediment	H/M
soil	M: in terms of exposure risk
non-local air sources (including deposition)	L
biota sinks	H

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	**Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)**	**Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)**	**Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)**
H	M	Y	
5	3 (upper quartile, as measured by serum, breast milk or adipose PCBs; depending on approach, could be a 5	2: from fish caught by persons living near contaminated estuaries	

	since that would be a quarter of the population)		
			H, 4

Human Health Issue Summary: PCBs

What are they?
There are more than 200 structurally similar polychlorinated biphenyls (PCBs) formerly manufactured for use in transformers and electrical components. They are chemically stable, which was a benefit in their industrial application, but has become a problem because of their persistence in the environment. While the use of PCBs was discontinued many years ago, they exist in the environment as a result of historic spills and disposal practices. Currently the greatest source of exposure to PCBs results from their presence in aquatic systems where they are taken up by aquatic organisms. Humans and wildlife may become exposed to PCBs through ingestion of food. PCBs are known carcinogens and cause developmental and reproductive problems in humans as well as several species of wildlife.

What's at risk?
PCBs bioaccumulate in the food chain. For humans, the primary exposure is via the ingestion of meat products. Consumers of large fish, particularly fish from areas with elevated concentrations of PCBs are the most likely to be exposed.

What are the human health impacts in New Jersey?
As many as 2,000 to 2,500 cases of cancer per year may be attributed to PCBs in New Jersey. This is approximately one third to one half of the total incidence of breast, pancreatic, and non-Hodgkins lymphatic malignancies in the state. There are, however, significant uncertainties in this assessment. There is also evidence that pre-and post-natal exposure to PCBs may have adverse effects on neurological development.

What's being done?
PCB production and use has been banned since 1977. Contaminated site clean up is taking place slowly and there are efforts to dredge contaminated sediments, including the large source that exists upstream on the Hudson River.

APPENDIX

In comparing the following population-based studies to the rat studies, it should be noted that the proportions of congeners grouped by degree of chlorination in studies in the Great Lakes approximate that in Aroclor 1260 (Anderson et al., 1998; Humphrey et al., 2000). Those proportions were similar in both fish-eaters and Acontrols@.

Breast cancer: Serum PCBs are linked to breast cancer in the upper tertile (>3.7 ng/g, range of 0.7-19 ng/g in all subjects from serum taken a decade earlier) among the 10-15% of women hetero- or homozygotic for a CYP1A1 polymorphism (Moysich et al., 1999). The adjusted OR = 2.9 (1.2-7.4). (These findings have been replicated in a cohort in Maryland, but the paper is in press.) A related polymorphism is found in African-American women and is strongly associated with breast cancer, regardless of PCB body burden. Its presence may be related to the finding by Krieger et al. (1994) of increased breast cancer risk (OR = 2.2 (95%CI, 0.7-7) in the highest tertile (>5 ng/mL). In the largest study of breast adipose tissue (Aronson et al. 2000) risk due to PCBs #105 and 118 (2,3,3',4,4' and 2,3,4,4',5) was increased among premenopausal women (ORs in the highest tertiles = 3.9 and 2.9, respectively), while in postmenopausal women a number of PCB congeners, particularly those with the highest concentration (hexa- and hepta-chlorinated PCBs #138, 153, 170, and 180) were associated with increased risk (ORs 1.5-3.3 for 6 of 10 and 7 of 10 measured PCBs in the middle and highest tertiles, respectively).

Non-Hodgkin's lymphomas (NHL): In a case-control study the incidence of NHL have also been linked to PCBs, but not DDT/DDE, Mirex, chlordanes, or hexachlorocyclohexane in serum collected 20 years earlier (Rothman et al., 1997). Among those with the highest serum concentration, 1070-2070 ng/g lipid (measured as 28 congeners), there was an adjusted (for DDT) OR of 4.1 (95%CI, 1.4-12). Conditional logistic regression that included socioeconomic factors and cigarette use yielded ORs of 2.9 (95% CI, 1.1-8.1) and 4.3 (95%CI, 1.6-12) for the third and fourth quartiles with a $p_{trend} = 0.003$. There was a significant positive interaction between seropositivity for Epstein-Barr virus (EBV) and PCB concentration. Among those with both exposures, there was an OR of 22 (95%CI, 4-115). The presence of EBV has been linked to NHL in some studies, and certain PCBs have been recognized as immunosuppressive, which is an established risk factor for NHL. 16 of 73 (22%) cases and 14 of 143 (10%) matched controls were seropositive. The causal relationship between EBV seropositivity, PCB exposure and cancer is not clear.

Notably, a study (Nordstr□m et al., 2000) of the rare hairy cell leukemia (HCL), related to NHL, found that among those with high EBV titers that immunosuppressive PCBs (#66, 105, 110, 118, 128/187, 138, 156, 170/190) above the median (>285 ng/g blood lipid) were highly associated with HCL (OR = 11, 95%CI 2-73). Among those with high EBV titers, total PCBs above and below the median were also significantly associated (OR = 4.4). (Levels of HCB, p,p=-DDE, and chlordanes above the median had ORs of 11, 6.6, and 15 among those with high titers.

A small case-control study found that PCB congeners with higher degree of chlorination (PCB #153 and over) were present at higher concentrations in adipose tissue of patients with non-Hodgkin=s lymphoma than non-malignant controls (Hardell et al., 1996). PCB congeners with less chlorination were mostly the same concentration in cases and controls, as were hexachlorobenzene, DDE, seven PCDDs and ten PCDFs. An OR of 2.7 (95%CI, 0.8-9.4) was observed for cases with the sum of PCBs greater than the median concentration of 1300 ng/g lipid no matter what the diagnostic staging.

Pancreatic cancer: Pancreatic cancer was associated with total serum PCBs in a case-control study (Hoppin et al., 2000) with a dose-response trend from lowest to highest tertile ($p<0.001$). In the highest tertile ($\geq$360 ng/g lipid) the OR was 4.2 (95%CI 1.8-9.4) compared with the lowest tertile. (The OR for p,p=-DDE was not elevated when adjusted for PCB levels.) Pancreatic cancer with a K-ras mutation has been strongly linked to serum PCBs in a small case-control study (Porta et al., 1999). Among those with a mutated K-ras oncogene (77% of cases and 17% of controls), odds ratios for pancreatic cancer in the highest tertiles (above approximately 0.25□g/g) for each of the three predominant congeners (adjusted by DDE level, alcohol, tobacco and coffee use) were 6.6 (95%CI 0.9-47, $p_{trend} = 0.05$) for PCB #138, 6.0 (95% CI 0.9-41, $p_{trend} = 0.07$) for #153, and 9.6 (95%CI 1.1-84, $p_{trend} = 0.03$) for #180. (Odds ratios for p,p=-DDT and p,p=-DDE, adjusted for PCB concentration and other exposures were 18 and 7.4, respectively.) Mean concentration of serum PCBs was about 1.8 times among cases with the mutations compared to wild type. There were no significant differences between cases with wild type K-ras and controls. The causal relationships between the mutation, PCB levels and cancer is unclear.

Neurodevelopment: There is evidence that prenatal and postnatal exposure to PCB mixtures and congeners in laboratory animals, as well as organochlorines measured in humans as PCBs, have detrimental effects on neurodevelopment (reviewed by Schantz, 1996). While all epidemiological studies have found associations between fish consumption history and/or PCBs in breast milk, maternal serum and cord blood, they have not been entirely consistent. Unfortunately, none of the studies measured the co-occurrence of mercury, which is also related to fish consumption. The North Carolina and Michigan cohorts have been followed the longest. Neonatal hypotonia and delayed infant psychomotor development (on Bayley Scales) was reported in North Carolina (Rogan et al., 1986; Gladen et al., 1988; Rogan and Gladen, 1991) and the Netherlands (Huisman et al., 1995a,b; Koopman-Esseboom, 1996), but not in Michigan, where neonatal hyporeflexia and negative effects on cognition and visual recognition memory (Fagan Test of Infant Intelligence) in infants were reported (Jacobson et al., 1984, 1985). (However, in the Michigan study, 5 months old may have been too early to make psychomotor assessments.) In 4 year olds in Michigan effects on IQ and short-term memory have been found (Jacobson et al., 1990a,b; 1992). In North Carolina the deleterious effects did not continue during testing at age 3-5 years or in report card grades at age 11 (Gladen and Rogan, 1991), while among children at age 11 in Michigan they did (Jacobson and Jacobson, 1996). It has been suggested that lower levels of PCBs and higher socioeconomic status may have masked deleterious effects in the North Carolina cohort. (The Dutch study measured all PCB congeners (though not all were detectable), and also measured PCDDs and PCDFs in breast milk.)

There are two other studies, which are at an earlier phase, having published only on neonatal neurodevelopmental measures. The German study (Winneke et al., 1999) found that Bayley mental development index was negatively correlated with PCBs in breast milk, but not the Bayley psychomotor development index and the Fagan Test. A study in Oswego

County, New York (on Lake Ontario) examined a population that included many with a diet significantly supplemented by sport fishing (Lonky et al., 1996). Newborns in the high exposure group scored more poorly on the reflex, autonomic, and habituation cluster of the Bayley Scales but not the orientation, range and regulation of state, or motor clusters.

The studies need to be replicated.

There is currently meager data linking PCDDs or PCDFs with retardation of neurodevelopment. The role of different types of PCB congeners is still being characterized.

Summary: Significant numbers of people appear to be affected by cancer and neurodevelopmental retardation associated with PCBs, but the mechanisms need to be better characterized and the studies need to be further replicated. The affect of other organochlorines needs to be further examined. There has been little analysis of PCB effects using the toxic equivalency approach.

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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Polycyclic Aromatic Hydrocarbons (PAHs)**
description of stressor (including etiology)	PAHs are compounds that contain only hydrogen and carbon present in two or more aromatic rings. The term "PAH" can be used interchangeably with the term "Polynuclear Aromatic Hydrocarbon" (PNA). PAHs are byproducts of incomplete combustion of organic material. They are ubiquitous in industrialized societies. Traces are found in air, food and water. Smoke or charcoal broiled foods are dietary sources with notably high levels of PAHs. Fugitive cigarette smoke is a major indoor source. Combustion products from motor vehicles, as well as from processes such the burning of fossil fuels and/or wood are other common sources of PAHs. The ubiquity of these materials in the environment and contributions from life style factors (such as smoking and smoked food consumption) are problematic confounding factors when trying to determine how much specific environmental sources contribute to body burden (ATSDR, 1993; Pike, 1992).
stressor-specific impacts considered including key impacts	Acute: All PAHs (both carcinogenic and non-carcinogenic) have a low order of acute toxicity in humans. Chronic: PAHs found in materials such as coal tar, for example, can produce a variety of non-cancer effects with chronic exposure. Chronic effects include the following: Eye irritation and/or photosensitivity Respiratory tract irritation with cough and bronchitis Dermal burns, irritation, erythema, photosensitivity, precancerous lesions such as coal tar warts (such lesions can be enhanced by UV light exposure). Mild hepatotoxicity or mild nephrotoxicity (observed in animals). Hematuria However, the most significant chronic toxicity endpoint for PAHs is cancer. Increased incidences of cancers of the skin, bladder, lung and possibly gastrointestinal tract cancers have been described in PAH-exposed workers, particularly associated with coal carbonization, coal gasification, and coke oven work. Again, not all PAHs are carcinogenic. PAHs with four or more fused rings tend to be carcinogenic while smaller PAHs tend not be carcinogenic. PAHs require metabolism to specific metabolites to cause cancer. Chronic exposure to PAHs may have an effect (positive/negative) on the ability of PAH to induce cancer (ATSDR, 1990, 1993).

Exposure Assessment	
exposure routes and pathways considered (include indoor air as appropriate)	Inhalation: Inhalation is a route of exposure to PAHs. PAHs are found in both outdoor and indoor air. In outdoor air, PAHs result from activities such as power and heat generation and refuse burning. Natural sources, such as forest fires, can also contribute to the atmospheric burden of PAHs. Sources of PAHs in indoor air include tobacco smoke, unvented space heaters, and food preparation. Inhalation is a major route of PAH exposure for smokers and for individuals in certain occupational settings e.g., coke oven workers, etc. (ATSDR, 1993; Holbrook, 1990). Ingestion: Ingestion is a major route of exposure to PAHs in non-smokers. PAHs can be found in foodstuffs due to biosynthesis, adsorption of particulates deposited on leafy surfaces from atmospheric sources, and processing or cooking of foods (e.g., charbroiling) before they are consumed (ATSDR, 1993; Pike, 1992). Dermal: Dermal exposure is typically a minor route of exposure. In general, exposure by this route is not well characterized and, therefore, is not currently considered by most regulatory agencies. Dermal exposure, however, can occur in certain situations and/or occupational settings e.g., use of coal tar shampoo for dandruff, coal tar treatment for psoriasis, exposure of auto mechanics to used motor oil, roofers (roofing tar), exposure to contaminated soil, etc. (ATSDR, 1993).
population(s)/ecosystem(s)exposed statewide	All New Jersey state residents have been and continue to be exposed to PAHs from multiple environmental sources including air, water, soil and food. However, the degree of exposure from these sources can vary significantly from region to region. In addition, individual lifestyle factors such as smoking and ingestion of smoked and charbroiled foods contribute to total body burden.
Quantification of exposure levels statewide (include indoor air as separate category as appropriate)	PAHs have been detected in the air, water, soils and plants of New Jersey. However, the data is very limited. The NJDEP has conducted several studies to evaluate the concentration of B(a)P in airborne particulate matter and several studies to measure the levels of selected PAHs in soil. **AIR:** In studies conducted between 1989 and 1995, mean levels of B(a)P in ug/m^3 ranged from 0.04 (Ringwood, NJ) to 0.621 (Newark, NJ). In general, levels tended to be higher in urban areas (NJ DEP, 1997, 1998). In their Cumulative Exposure Project (CEP), the US EPA estimated that in 1990 NJ had a mean statewide air concentration of 0.49 ug/m3 for polycyclic organic matter (POM). This value is in the same range found in the studies above. Of note, these values vary by an order of magnitude. **Indoor Air**: Indoor air concentrations of BaP were measured in homes in Phillipsburg, NJ. Estimates of inhalation exposures to BaP among the study subjects ranged from 10 to 50 nanograms/day. Sources of indoor levels of BaP included smoking, cooking, wood burning and penetration of outdoor air indoors. In most cases, indoor air BaP levels were closely correlated with ambient levels. (Lioy and Greenberg, 1990; Waldman and Lioy et al., 1991). **SOIL:** Selected PAHs were measured in urban surface soils in 1997 (Piedmont Region) and 1998 (Coastal Plain Region). The median extractable concentrations of selected PAHs (mg/kg) from soil in each region are listed below: (NJ DEP, 1989-1995). Of note, all of these values are below the NJDEP soil cleanup standards for NJ.

<table>
<tr><td></td><td>
<table>
<tr><td>PAH</td><td>Piedmont
[Median] mg/kg</td><td>Coastal Plain
[Median] mg/kg</td></tr>
<tr><td>Benzo(a)anthracene</td><td>0.21</td><td>0.04</td></tr>
<tr><td>Benzo(a)pyrene</td><td>0.21</td><td>0.02</td></tr>
<tr><td>Benzo(b)fluoranthene</td><td>0.23</td><td>0.19</td></tr>
<tr><td>Benzo(k)fluoranthene</td><td>0.22</td><td>0.19</td></tr>
<tr><td>Dibenz(a,h)anthracene</td><td>0.21</td><td>0.01</td></tr>
<tr><td>Indeno(1,2,3-cd)pyrene</td><td>0.20</td><td>0.01</td></tr>
<tr><td>Chrysene</td><td>-</td><td>0.05</td></tr>
</table>
It should be noted that considerable uncertainty exists in the PAH concentrations listed above. Most of the results are between the limit of detection and the practical quantitation level for these contaminants. Furthermore, the Piedmont and Coastal Plain sample sets were conducted by different laboratories, so the general difference observed in the results from the 2 data sets may be partly attributable to systematic differences between the laboratories. Dibenz(a,h)anthracene was undetected in most samples in both the urban Piedmont and urban Coastal Plain studies. Since DEP policy in these cases is to set the concentration at one-half the detection limit, the median value is largely determined by this value, rather than by actual observed concentrations. In the Coastal Plain study, the remaining PAHs were detected in only one-third to one-half of the samples, and the median value is again determined to be at one-half the detection limit for these contaminants.</td></tr>
<tr><td>specific population(s) at increased risk</td><td>Higher relative risk populations include workers in certain occupations that have elevated PAH concentrations in the ambient work environment such as roofers, and coke oven workers. In addition, smokers and individuals living near industries such as creosote and coal tar manufacturers, that generate PAHs as a byproduct of production, are also at higher relative risk (ATSDR, 1993).

Individuals in these higher relative risk exposure groups may have varying susceptibilities to PAH-induced diseases. For example, increased cancers have been observed in coke oven workers etc. (IARC, 1987; Ellenhorn & Barceloux, 1988; ATSDR, 1990, 1993;</td></tr>
<tr><td>Quantification of exposure levels to population(s) at increased risk (due to factors other than exposure)
(include indoor air as separate category as appropriate)</td><td>PAHs require metabolic activation to be carcinogenic. Factors that alter metabolism (increase or decrease it) will impact the biological effects of PAHs. Populations having certain metabolizing capacities may be more susceptible to PAH-induced cancers. Studies have also demonstrated that young animals are more susceptible to the effects of PAHs. Therefore, the younger population may also be at relatively increased risk (Calabrese, 1984; EPA, 1980, ATSDR, 1993).</td></tr>
<tr><td>Dose/Impact-Response Assessment</td><td></td></tr>
</table>

quantitative dose-impact-assessment employed for each population considered	Estimates of body doses or tissue levels associated with exposure to PAHs such as BaP are highly uncertain. In order to develop such estimates, the following information is required: Chemical concentration of the PAH(s) of concern in environmental media (e.g., soil, air, water, food, etc.) Exposure assumptions (e.g., 100 mg soil ingested/day; 20 m^3 air inhaled/day Information on the absorption (bioavailability) of the PAH(s) from matrices such as soil or particulate matter, etc. The extent of absorption will be variable and dependent on the composition of the matrix. B(a)P has typically been used as an indicator compound for a mixture of PAHs. Most, but not all PAHs, are less active than B(a)P. However, mixtures may be more or less active than the sum of their constituents. Mixture effects are a major data gap for all environmental contaminants. The EPA has developed a scale of the relative carcinogenic potency of PAHs relative to B(a)P. The relative carcinogenic potencies of the known carcinogenic PAHs are (Thorslund et al., 1986): Benzo(a)pyrene: 1.0 Benz(a)anthracene: 0.1 Benzo(b)fluoranthene: 0.1 Benzo(k)fluoranthene: 0.01 Chrysene/triphenylene 0.001 Dibenz(a,h)anthracene: 1.0 Indeno(1,2,3,cd)pyrene: 0.1 The oral slope factor for B(a)P is 7.3×10^{0} per (mg/kg)/day (or 7.3 excess deaths per mg/kg ingested/day). The drinking water unit risk is 2.1×10^{-4} per (μg/L) or 2.1 excess deaths per 10,000 per μg/L drinking water ingested (IRIS Database, 2000).
Risk Characterization risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	A NJ-specific risk characterization for PAHs is beyond the scope of this document. Quantitative risk characterization requires good exposure data. NJ residents may be exposed to multiple PAHs from several media sources (e.g. air, soil) by several routes of exposure (e.g., inhalation and ingestion). The available exposure data is incomplete. Consequently, the risks associated with exposure to PAHs in the NJ environment cannot be accurately determined at this time.

assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	PAHs are absorbed following ingestion, inhalation and dermal exposure. Following absorption, PAHs can enter the lymph and then the blood stream. PAHs are metabolized primarily in the liver by the Cytochrome P-450 enzyme system. Metabolism in non-hepatic tissues e.g. kidneys, has also been demonstrated. Many PAHs are transformed into non-toxic polar metabolites that are efficiently excreted in the urine and bile. The majority of PAH metabolites are phenols, diols and quinones. In general, further metabolism of diols to diol-epoxides results in the formation of reactive metabolites that are responsible for the carcinogenic properties of PAH. For example, the 7,8-dihydro-7,8-diol-9,10-epoxide is considered to be the ultimate carcinogenic metabolite of benzo(a)pyrene. Diol epoxides bind to DNA and other cellular components to initiate the carcinogenic process (Pike, 1992; ATSDR, 1993; Amin, 1993; Ronai et al., 1994).
size of population(s) affected	As PAHs are ubiquitous in the environment, all residents of NJ would be exposed to some extent.
assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	H As PAHs are so ubiquitous, it is difficult to determine with any degree of certainty those environmental sources that are responsible for increased cancer risk.
potential for future changes in the underlying risk from this stressor, (+++, ++, +, 0, !, =,/ where + is improvement)	++ Reformulation of fuels (e.g., lowering sulfur content of diesel fuel) may reduce PAH emissions from diesel vehicles; stricter programs for inspecting emissions from motor vehicles, etc.
potential impact from catastrophic (low probability) events (H, M, L) and brief description of likelihood	L
extent to which risks are currently reduced through in-place regulations and controls	M (e.g., decreased emissions from industrial facilities, mandatory industrial waste site cleanups, use of protective clothing/gloves)
Relative Contributions of Sources to Risk (H, M, L)	

Allocation of stressor-specific risk to primary NJ sources	
large business/industry	L- M (e.g., coke facilities, coal burning power plants, oil refining operations, etc.) Of note, emissions would only be high in areas close to these industrial sources. Thus, there would be significant differences in different parts of the state.
small business industry	M (e.g., roofing contractors, restaurants)
Transportation	H (diesel vehicles, railroads)
Residential	M (e.g., wood burning, char-broiling foods, smoking)
Agriculture	L – virtually no burning of agricultural fields in NJ
Recreation	L
resource extraction	L
Government	M (e.g., military sites - jet fuel, fuel oils, diesel powered vehicles etc.)
Natural sources	L/M (e.g., forest fires, wood burning stoves)
Contaminated sites	M/H
Diffuse and non-NJ sources	
Sediment	M
Soil	M
non-local air sources (including deposition)	M-H (e.g., near-by industrial facilities) The NJ Atmospheric Deposition Network (NJADN) obtained atmospheric data on selected PAHs. Based on this data it can be concluded that some PAHs have higher summer concentrations while others have higher winter concentrations. Thus, the atmospheric concentration of individual PAHs may vary depending upon the season and wind direction. In most cases, the origin of atmospheric PAHs (i.e., local vs. non-local sources) cannot be easily determined.
biota sinks	L (PAHs do not bioaccumulate or bioconcentrate)

Issue: Polycyclic Aromatic Hydrocarbons (PAHs)
Author: Deb Edwards, Marie Amoruse
Version: 08/01

What is it?
Polycyclic Aromatic Hydrocarbons (PAHs) are chemical compounds containing hydrogen and carbon that result from the incomplete burning of organic material such as cigarettes, wood, food, and fossil fuels. PAHs are found nearly everywhere in the environment, both naturally and as a result of human activities. There are thousands of PAHs; of particular concern are those that cause cancers, including skin, bladder, lung, and possibly the gastrointestinal tract. Other effects of long term exposure may include eye irritation and light sensitivity. Exposure to PAHs may occur via inhalation, ingestion of smoked or charbroiled foods, or as a result of skin contact with coal tars in shampoos and psoriasis treatments.

What's at risk?
All New Jersey residents have been and continue to be exposed to PAHs, however, the degree of exposure from these sources can vary greatly from region to region, with higher levels in urban areas. In addition, personal lifestyle choices such as smoking and ingestion of smoked and charbroiled foods contribute to an individual's body burden. PAHs must be acted upon by the body's metabolic processes in order to become carcinogenic. Children and adolescents may be at increased risk due to higher rates of metabolism. Additional groups at risk include roofers and coke oven workers, and individuals living near creosote and coal tar manufacturers.

What are the human health impacts in New Jersey?
There are insufficient exposure data available to quantify the number of illnesses in New Jersey.

What's being done?
Emissions from industrial facilities are regulated, industrial hazardous waste sites are undergoing mandatory cleanup, and protective clothing is used in occupational settings to reduce risk.

{PRIVATE } **Severity of specified health effects at current levels of exposure**{PRIVATE } **(H,M,L)** **(also 1-5 with 1 being least severe)**	**Size of population at significant risk for each health effect (H,M,L)** **(also 1-5 with 1 being smallest)**	**Are there discreter communities at elevated risk?** **(Y,N)** **(also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)**	**Overall risk ranking (as a function of severity and population effected integrating across health effect)** **(H,M,L)** **(also 1-5 with 1 being the lowest overall risk)**
2	M	Y/N (urban areas)	2

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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Sressor	**Radionuclides** Radiological releases resulting from the routine operations of a nuclear power plant. Radium, and radon are dealt with in separate assessments
description of stressor (including etiology)	The operating license issued to each nuclear power plant includes technical specifications that prescribe limiting conditions for the daily operation of the plant. The U.S. Nuclear Regulatory Commission (NRC) establishes limits for the release of radioactivity during routine operations. NRC-licensed nuclear power plant operators are required to monitor daily effluent emissions in accordance with the NRC's 10CFR20, Appendix B and comply with standards prescribed in 10CFR50, Appendix 1. The limits to radioactive releases are set at 10 millirem/year for air and 5 millirem/year for water. As part of the normal functioning of a nuclear plant, some releases do occur that result in slightly radioactive gaseous discharges to the environment. These are considered a normal function of the reactor's operation as long as they do not exceed the limits regarding the effluent permitted to be discharged offsite in 10CFR Part 50, Appendix I. The NJDEP's Bureau of Nuclear Engineering (BNE) operates an Environmental Surveillance and Monitoring Program (ESMP) that independently monitors and assesses radiation in the environment outside the site boundaries of New Jersey's four nuclear generating stations. The data obtained is used to determine the affect, if any, of the operation of the plants on the environment and human health. Part of the ESMP includes air monitoring and analysis of specific gamma and gross beta radionuclides in the immediate vicinity of the plant sites. As part of normal operations, the plants may release the following radionuclides: krypton; xenon; iodine; tritium; cesium; cobalt; strontium; antimony; manganese; yttrium; zinc; technetium; chromium; zirconium; iron; barium. Of the specific gamma radionuclides, iodine-131 is of particular interest because it is the most abundant radionuclide measured at a nuclear power plant. Iodine-131 data collected by the BNE over many years show average radionuclide in air measurements many orders of magnitude below NRC's limits in 10CFR20, Appendix B. On a national scale, the Committee on the Biological Effects of Ionizing Radiation (BEIR), of the National Research Council, compiled data on the exposure of individuals to radioactivity. The BEIR V data shows that radiation from occupational activities, nuclear power production (the fuel cycle), and miscellaneous environmental sources (including nuclear weapons testing fallout) contributes negligibly to the average effective dose equivalent received by the general public, i.e., less than 0.01 millisievert/year (< 1 millirem/year).
	See Appendix, Figure 1, “Source of Radiation Exposure to the U.S. Population,” which is taken from page 19 of Health Effects of Exposure to Low Levels of Ionizing Radiation, BEIR V, National Academy Press, 1990.

stressor-specific impacts considered including key impacts	Radiation exposure from both natural and made-made sources is most notably associated with induction of cancer. Other health effects include genetically associated disorders, developmental abnormalities and some degenerative diseases. (BEIR V Executive Summary, page 1) A September 1990 National Cancer Institute (NCI) study found no evidence of any increase in cancer mortality including childhood leukemia among people living in 107 counties that host, or are adjacent to, 62 major nuclear facilities in the United States. The conclusions of the NCI study, the broadest ever conducted, are supported by many other scientific studies in the United States, Canada and Europe. (See "Risk Characterization" for a more detailed discussion of the study.)
Exposure Assessment	
exposure routes and pathways considered **(include indoor air as appropriate)**	Radionuclides are released from nuclear generating stations during routine operation through gaseous and liquid effluents. If large quantities were released, the airborne radionuclides could be inhaled or deposited in the environment and ingested through consumption of vegetation, milk or water. The allowable release limits are specified in NRC's 10CFR Part 50 and may not be exceeded. Data collected by the Bureau of Nuclear Engineering's environmental monitoring program shows the ambient concentrations of radionuclides around the plant sites to be many order of magnitudes below NRC's allowable limits, and not significantly different than normal background radiation levels.
population(s)/ecosystem(s) exposed statewide	Because of the nature of radioactive deposition and decay, the populations at greatest risk of exposure to radionuclides from nuclear generating stations are those closest to the plant site. Oyster Creek Nuclear Generating Station is located in Lacey Township, Ocean County. Salem 1, Salem 2 and Hope Creek are sited at Artificial Island on the Delaware River in Salem County.
quantification of exposure levels statewide, including populations at significantly increased exposure **(include indoor air as separate category as**	The NRC designates two zones around each plant site to express potential risk to the population. (See Appendix Figure 2, "Nuclear Power Plants Affecting the State of New Jersey".) Ten miles around each plant site is designated as the "Emergency Planning Zone" or EPZ. Should a nuclear event occur at a plant, procedures are delineated to

appropriate)	evacuate or shelter these populations. All those residing in this zone are notified annually of the procedures in place and their necessary response. The population of the combined EPZs for both Oyster Creek and Artificial Island is approximately 288,000, according to State Police's Office of Emergency Management. Fifty miles around each plant site is designated as the "Ingestion Pathway Zone" or IPZ. Should a nuclear release in excess of NRC's allowable limits occur, the radionuclide deposition would most probably affect this zone. All vegetation growing in the IPZ potentially could be contaminated by the deposition. The foodstuffs/vegetation could be ingested by humans directly or by livestock and then by humans in turn. Because of this, plans exist to quarantine the agricultural products of the affected zone should a release of radioactive gaseous effluent in excess of allowable limits occur. The population of the IPZ is difficult to estimate because it extends into several neighboring states, including Delaware, Pennsylvania and Maryland, and because it includes population within the IPZ of plants in neighboring states. However, based on the figure, most of the population of NJ is within the IPZ of one or more plants.
specific population(s) at increased risk	Children and developing fetuses are at greatest risk from exposure to any form of radiation. However, the Bureau of Nuclear Engineering's environmental monitoring data indicates there are no elevated levels of radionuclides from the power plants in the environment around Oyster Creek and Artificial Island.
quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) **(include indoor air as separate category as appropriate)**	Data is not available from State Police's Office of Emergency Management to quantify the number of children and pregnant women in the Emergency Planning Zones around Oyster Creek and Artificial Island. To put in perspective the amount of radiation to which these populations are exposed, the NCRP87b study attributes only 0.1% of all sources of radiation exposure to the U.S. population as coming from the nuclear fuel cycle (i.e. operation of nuclear generating stations.) The majority of exposure comes from radon (55%), with cosmic, terrestrial, internal, medical x-rays, nuclear medicine and consumer products together contributing almost all of the remaining 45%.
Dose/Impact-Response A**ssessment**	The quantitative relationship between cancer incidence and dose in atomic bomb survivors, as in other irradiated populations, appears to vary, depending on the type of cancer in question. The dose-dependent excess of mortality from all cancer other than leukemia shows no departure from linearity in the range below 4 sievert, whereas the mortality data for leukemia are compatible with a linear-quadratic dose response relationship. (BEIR V, Executive Summary, page 5) Of the various types of biomedical effects that may result from irradiation at low doses and low dose rates, alterations of genes and chromosomes remain the best documented. In spite of the evidence that the molecular lesions which give rise to somatic and genetic damage can be repaired to a considerable degree, the new data do not contradict the hypothesis, at least with respect to cancer induction and hereditary genetic effects, that the frequency of such effects increases with low-level radiation as a linear, nonthreshold function of the dose. (BEIR V, Executive Summary, page 4.)
quantitative dose/impact-assessment employed for each	

population considered	
Risk Characterization	
risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	A study conducted by the National Cancer Institute (NCI) and published in the Journal of the American Medical Association, March 20,1991, showed no general increased risk of death from cancer for people living in 107 U.S. counties containing or closely adjacent to 62 nuclear facilities. The facilities in the survey had all begun operation before 1982. The survey examined deaths form 16 types of cancer, including leukemia. In the counties with nuclear facilities, cancer death rates before and after startup of the facilities were compared with cancer rates in 292 similar counties without nuclear facilities. The study also reported radioactive releases from monitored emissions of nuclear facilities in the United States show very low radiation exposure to the surrounding populations. Maximum individual radiation doses from these plants are reported to be less than 5 millirem annually, or less than 5 percent of what is received annually from natural background sources of radiation, such as cosmic rays and radon. Levels this low are believed to be too small to result in detectable harm. It should be noted that Ocean and Salem counties in New Jersey were part of this NCI study. Therefore, the conclusion of the study can be directly applied to the populations surrounding Oyster Creek and Salem.
assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	***The severity, persistence, irreversibility of cancer is considered to be high, however, the frequency of effects are all very low to non-existent for the risks associated with normal, ongoing releases of radionuclides from nuclear generating stations***
size of population(s) affected	As stated under "Exposure Assessment", the combined population in the Emergency Planning Zones (ten-mile radius of each plant site) of both Oyster Creek and Artificial Island is approximately 288,000.
assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	(L) There is a low level of uncertainty in this assessment, as data consistently bears out that extremely low levels of radionuclides are emitted from the nuclear generating stations and there is low risk associated with such low doses.
potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	(L)
potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	(0) There is always the possibility that through aging there will be increased degradation of plant equipment and the potential for increased release of radioactive emissions. The corrosion of the reactor vessel head from borrated water at Davis Besse in Ohio and the cracks in the reactor nozzles at Oconee in South Carolina suggest this as a

	possibility. However, this is mitigated by mandated inspection and replacement of defective equipment. However, this risk is mitigated by regular and mandatory inspections and replacement of defective equipment.
potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	(L) The risk of a catastrophic event occurring at a nuclear generating station in NJ is extremely low. The associated impact, of course, would be far reaching. To put in perspective the risk of such an event occurring, consider that the accident at Three-Mile Island (TMI) was the only General Emergency ever declared at a nuclear power plant in the United States. It involved a relatively small release of radionuclides. Chernobyl, a far more serious accident, released significant amounts of radionuclides. BEIR V provides these comments on the effects of TMI and Chernobyl: It is still too early to assess whether any cancer excess will occur following the Three Mile Island or Chernobyl nuclear reactor accidents. The collective dose equivalent resulting from the radioactivity released in the Three Mile Island accident was so low that the estimated number of excess cancer cases to be expected, if any were to occur, would be negligible and undetectable (Fa81). For the Chernobyl accident, preliminary estimates suggest that up to 10,000 excess cancer deaths could occur over the next 70 years among the 75 million Soviet citizens exposed to the radioactivity released during the accident, against the background of 9.5 million cases of cancer that would occur spontaneously; hence the excess would not be detectable. However, among the 116,000 people evacuated from immediate high-exposure areas in the Ukraine and Byelorussia, there might be a detectable increase in the cases of leukemia and solid cancer (An88, No86.) ***It should be noted that an event such as occurred at Chernobyl could not be repeated in the United States because of the inherent differences in reactor types and design. A series of operator errors directly causing the Chernobyl incident, coupled with poor safety systems and a deficient containment structure, lead to the magnitude of the accident. The nuclear industry has learned tremendous lessons from both Chernobyl and Three Mile Island, and the resulting procedures, training and safety features now incorporated at all US plants greatly reduces the risk of any type of nuclear incident at a commercial generating station.***
extent to which risks are currently reduced through in-place regulations and controls	NRC's license requirements mandate radiation levels for all activities must be "As Low As Reasonably Achievable" (ALARA). The NRC defines ALARA as follows: Making every reasonable effort to maintain exposures to ionizing radiation as far below the dose limits as practical, consistent with the purpose for which the licensed activity is undertaken, taking into account the state of technology, the economics of improvements in relation to state of technology, the economics of improvements in relation to benefits to the public health and safety, and other societal and socioeconomic considerations, and in relation to utilization of nuclear energy and licensed materials in the public interest (see 10 CFR 20.1003). Regulation 10CFR50 Appendix I limits releases from nuclear power reactors to an exposure level of 5 millirem from liquid effluents and 10 millirad due to gamma radiation from gaseous effluent.

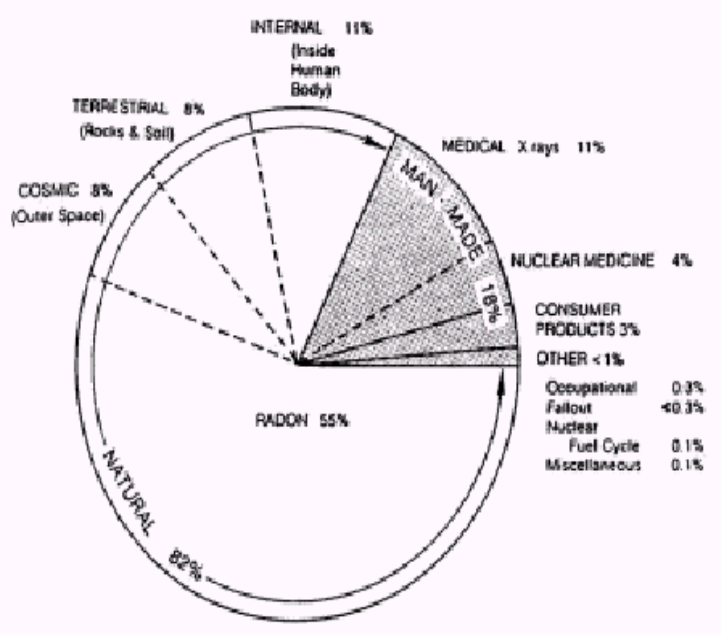
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
large business/industry	All State wide **L**
small business industry	All State wide **L**
transportation	All State wide **L**
residential	All State wide **L**
agriculture	All State wide **L**
recreation	All State wide **L**
resource extraction	All State wide **L**
government	All State wide **L**
natural sources	All State wide **L**
contaminated sites	All State wide **L**
diffuse and non-NJ sources	
sediment	All State wide **L**
soil	All State wide **L**
non-local air sources (including deposition)	
biota sinks	

verity of specified health effects at current levels of exposure (H,M,L)	**Size of population at significant risk for each health effect**	**Are there discrete communities at elevated risk?**	**Overall risk ranking (as a function of severity and population effected**

(also 1-5 with 1 being least severe)	**(H,M,L) (also 1-5 with 1 being smallest)**	**(Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)**	**integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)**
L	H	Y	L
5	5	1	1
			L,1

APPENDIX

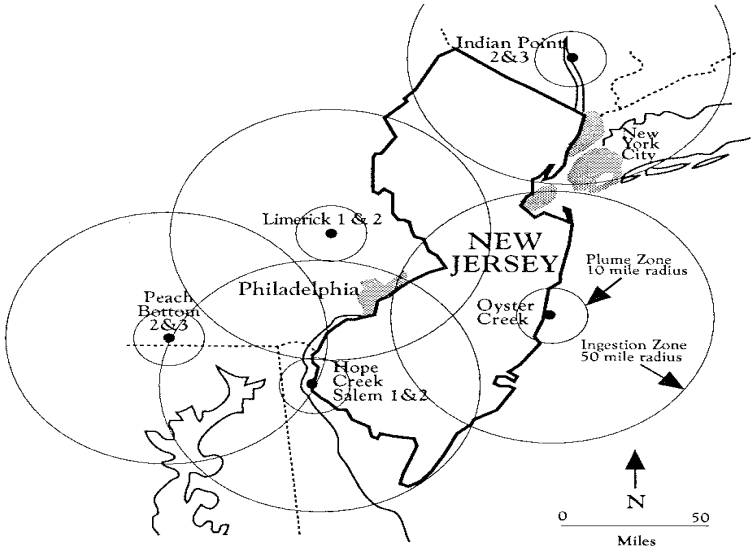
Figure 1



Source of Radiation exposure to the U.S. population (NCRP87b).

Figure 2

Nuclear Power Plants Affecting the State of New Jersey



New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor:	**Radium**
Description of stressor (including etiology)	Radium is a naturally occurring radioactive element, with three major isotopes: 226Ra, 228Ra, and 224Ra. The isotopes are derived from radioactive decay of precursor elements (Uranium 238 decays to radium-226; the other radium isotopes are derived from the decay of thorium 232). Radium isotopes vary in radioactive half-lives: 226Ra, 1,622 years; 228Ra, 5.75 years; 224Ra, 3.6 days. 226Ra decays to radon. The Environmental Protection Agency (EPA) maximum contaminant level (MCL) for combined 226Ra and 228Ra is 5 picocuries per liter of water (5pCi/L). Canada's target concentration for radium is 100 mBq/L, or about 3 pCi/L. There are no U.S. federal or state standards for 224Ra. The EPA MCL for gross alpha radioactivity in drinking water is 15 pCi/L. Radium is commonly present in rocks, soil and water in parts of New Jersey and neighboring states due to natural hydro-geological conditions and formations. Radium is chemically similar to calcium, is absorbed by plants through soil, and passes through the food chain to humans. Between 5% and 20% of ingested radium is absorbed, and 90% of the absorbed material is concentrated in bones. Residual radium has been measured in the body 20 years after exposure. Radium passes through mother's milk to the feeding infant; it also crosses the placenta during pregnancy, and is retained in fetal bones.
Stressor-specific impacts considered, including key impacts	The risks associated with radium isotopes are primarily due to radioactivity. Increased occupational radium exposure has produced human bone sarcomas and cancers of the perinasal sinus and air cells of the head (1-7). Increased total and acute myeloid leukemia has been reported in human ecological and occupational health studies, but the evidence is inconsistent (8). Other toxicity reported from high levels of exposure include cataracts, anemia, tooth and bone fractures, and general deterioration of bone tissue. The health risks of lower levels of exposure, such as may occur from drinking water exposures, have been studied in several general populations in the United States and Canada. The Canadian studies found an increased risk of bone sarcoma associated with the presence of radium in birthplace water, but no dose-response trend (9). Small increases of bone cancer were also noted in community water supply studies in Iowa and Illinois (10,11). A concentration-disease relationship between leukemia incidence and radium in well water samples was noted in Florida and suggested in Germany (8,12). The cancer risks associated with low-level radium consumption are difficult to assess and are still not adequately understood.

Exposure Assessment	

Exposure routes and pathways considered	The primary source of exposure to the general population to radium is through ingestion of drinking water. Between 0.1 and 1.0 Pci/L of total radiation is present in most natural water (13). Radium in rocks and soil readily dissolves in acidic water. In New Jersey, radioactivity is elevated in water derived from sections of the Kirkwood-Cohansey Aquifer system in southern counties and from groundwater passing through mudstone and sandstone formations of the Newark Basin in north-central counties (14,15). Radium also exists in plants and can be delivered to humans through the food chain. In some areas of the United States, food is a greater source of exposure than is drinking water. The radium content of the total daily diet is about 40 to 50 mBq/day (or about 1.1 to 1.4 pCi/day), resulting in internal radiation dose of about one mrem per year (16;16a). Some foods, such as Brazil nuts, have radium concentrations up to 1000 times that of the average dietary composition (17). High occupational exposures resulted from radium processing and luminescent paint application in New Jersey companies of the early 20th century. Contamination of workplace sites and proximate neighborhoods have occurred. Several of these New Jersey sites are on the EPA National Priority Lists and have been the subject of Superfund-driven remediation programs (18,19,20).
Population(s)/ecosystem(s) exposed statewide	Drinking water: Southern New Jersey The shallow, unconfined aquifers of the Kirkwood-Cohansey system provide the majority of private well drinking water for about 500,000 south New Jersey residents (21). Analyses of 170 wells (22) served by this system reveal that over 30%, including 21% of public supply wells, exceed the combined 226Ra and 228Ra MCL of 5 pCi/L. The majority of wells (52%) from both the agricultural and Bridgeton Formation outcrop areas exceed the MCL. In addition, the gross alpha radiation standard of 15 pCi/L was surpassed in nearly 20% of water samples obtained throughout the Kirkwood-Cohansey aquifer system. Recent studies have also identified 224Ra in samples (23). Prior standard analyses were performed one month after collection, and therefore could not have detected the short- half-life isotope. No standards currently exist for the 224Ra isotope. Drinking water: North-Central New Jersey The Newark Basin covers ten New Jersey counties, 2,000 square miles, and is the primary water source to more than five million state residents. The arkosic sandstones of the Stockton Formation and the black mudstones of the Passaic Formation are the major sources of radiation for water in the Basin.
	Water from 40% of ten Basin observation wells of North-Central New Jersey contains elevated gross alpha activity (over 15 pCi/L), and 20% of those wells also exceed the combined 226Ra and 228Ra standard of 5 pCi/L (15). Increased beta activity was seen in 3 of the 10 wells sampled.

	While the Newark Basin studies were of test wells, not drinking water supplies, they indicate the high potential for increased radioactivity in the Basin's interconnected, geologically fractured, regional water network. Most water consumed in the Newark Basin region is derived from surface water, which has low radium potential. Suppliers such as Elizabethtown Water Company obtain more than 90% of water from surface sources, with only about 10 % coming from wells (24). *Site contamination*: New Jersey EPA National Priority List sites with known radium contamination are isolated from the public and have been or are being remediated. However, gross radium contamination of soil, buildings, and covered surfaces has been identified (18,19,20). Public exposures occurred from some of these sites in the past. There are also over a dozen radium-contaminated sites in NJ that are not on the NPL. These sites are being remediated through the Industrial Site Recovery Act.
Quantification of exposure levels statewide	Approximately 5.5 million people are serviced through private wells or public supplies obtaining water from the K-C aquifer or the Newark Basin. Radiation and other contaminant concentrations are reduced in public water supplies through admixtures from multiple water sources. Radioactivity levels can also be greatly reduced by water softening treatment at the receiving end, but estimates of actual use of water softeners in New Jersey for the water supplies of interest have currently not been identified. Therefore, it is difficult to estimate actual water concentrations and population intake of radium isotopes in the state. If 20 to 30% of the tested water samples were found to exceed the MCL, and if 80 to 90% of that water were further rendered acceptable through dilution or treatment, then one could estimate population over-exposures. It is estimated that between 100,000 to 300,000 individuals in the noted state areas exceed the radium drinking water standards. This number could be higher or lower. Several hundred people may have previously been exposed to radium contamination from New Jersey Superfund sites. Except for hazardous waste workers, there should be few current public exposures from those sites. Data on food radium content, and potential daily intake via food consumption, were not identified for crops and dairy production derived from the agricultural section of southern New Jersey. Such information is important to scope potential aggregate exposure and public health risks more fully.
Specific population(s) at increased risk	Such populations include those with private well water, unmonitored for radiation; those in zones of highest radium content; and communities with high radium concentrations in public water supplies. Others of increased risks include individuals with high intake of water (e.g. children, diabetics, manual laborers, etc.) or increased bone metabolism, for example such disorders as Pagets disease. 224Ra measurement data are not available for much of the state, and could identify specific populations at increased risk. The fetus tends to concentrate radium in bone, and could present increased risk.

Quantification of exposure levels to population(s) at increased risk	Moderately high. Radium concentrations have been found in some water supplies at over four times the MCL. These supplies have been the primary source of drinking water for communities and families – and have likely been contaminated for the duration of human habitation on the sites. Replacement of contaminated private well water with community or bottled water eliminates water-borne exposures. Water softening at the receptor end reduces radium activity 90% or more (25).
Dose/Impact-Response Assessment	The impact for radium (particularly 224Ra) increases for a given dose as the time of irradiation is stretched over longer dosing periods (26). Thus, long-term exposure studies are particularly relevant to drinking water dose-response estimates. Metabolic models for absorption, radium storage and cumulative activity (27) in bone have been recently applied in assessing dose-response from radium intake via drinking water in Canada (28). While increased odds ratios of 1.38 (95% CI, 1.08-1.73) for all sarcomas, and 1.44 (1.01-1.87) for osteosarcoma were found for geometric mean doses to the bone of 26 mRad, there was no trend with increasing dose observed. The results of this study are compatible with no increased risk at low dose, but just as well could be explained away on the basis of low statistical power at the lower dose levels. Data from dose-response (bone sarcoma death) studies of radium dial painters (3), 234Ra injection (4), and from earlier Public Health Service investigations of drinking water studies, all fit a linear regression line of Rad dose versus risk (28). The regression equation of risk of fatal bone sarcoma per 100,000 people per year is 1.81 X dose in Rad. Increased risks of cranial sinus tumors have been observed with internal irradiation sources of 226Ra only – not with 228Ra or 224Ra (26). 226Ra produces radon 222 as a daughter product, and radon 222 accumulates in the sinuses, causing mucosal lining irradiation. The other radium isotopes do not produce radon 222 as a daughter product, and therefore are not likely to be cranial sinus risk factors. Annual risk has been estimated at 16 excess sinus tumors per million persons per 1 uCi of 226Ra (29). Most governmental agencies assume that radiogenic risk for bone malignancy is a linear non- threshold risk, although some have suggested that radium dial worker study data is compatible with a threshold (30). Dose for different types of radium is delivered to different portions of bone, based on the metabolic cycle of bone. 226Ra decays mainly in the bone volume, while 224Ra has a higher endosteal dose potential. Thus, judgements on the shape of the dose-response curve may well be influenced by local bone dynamics and radiation source type.
Quantitative dose/impact-assessment employed	EPA's most recent cancer risk coefficients for 226Ra, 224Ra, and 228Ra have been developed for mortality and morbidity associated with water intake (31). Assumed lifetime consumption of 1.11 liters of water daily, over a 75.2year time frame, results in 30,000 liters. The EPA cancer morbidity coefficient per Bq of 226Ra in drinking water is 1.04E-08, and for a lifetime consumption of 30,000 liters presents a risk of 3.12E-4. The risk per pCi content of drinking water would be $1/27^{th}$ of that per Bq, or about 1.15 E-5 lifetime risk. EPA considers the risk to be linear and with no apparent threshold. There is some counter information regarding possible hormetic, protective effects at low levels of radiation (32), but the evidence to date is only suggestive and not currently adequate to influence public policy.

Risk Characterization	
Risk estimate(s) by population at risk, including probability and number of cases/occurrences (specific metric employed, e.g., mean population risk upper percentile population risk, etc.)	Using the EPA cancer risk coefficients, the radium level of radioactivity at which the cancer morbidity risk is less than one additional case in one million people exposed in drinking water for a lifetime corresponds to about 0.1 pCi/L. That level is below generally regarded background values. One additional cancer case per 100,000 people per lifetime would be expected to result from about 1 pCi/L of 226Ra. Risks are similar for other key isotopes, about three times higher for 228Ra, and about 50% less for 224Ra. Applying the 226Ra risk as an approximation for total combined radium isotopes of 226Ra and 228Ra, we can roughly estimate the number of excess cancer cases predicted for New Jersey consumers of radium-containing water. We have generally estimated that there may be between 100,000 and 300,000 individuals with drinking water exceeding 5 pCi/L of combined radium. We estimate, based upon the general shape of the distribution of available well sampling data, that the average radium content for this tail of the distribution (i.e., values above 5 pCi/L) is likely to approximate 7 pCi/L. Thus, at 7 excess cases per 100,000 people at risk per lifetime, we estimate that between 7 and 21 excess cancers could occur in the population drinking water exceeding the combined radium MCL. Of course, the risk uncertainty is considerable, and the excess could be as low as zero or could be higher than the numbers stated. A substantial number of additional cases might also be estimated from the large population using water supplies with lower combined radium values, between 0.1 and 4.9 pCi/L. Also, additional exposed populations can be identified from total alpha data. It has been roughly estimated that as many as 125,000 to 150,000 people exceed the 15 pCi/L MCL. While many of those would be included in the group also exceeding combined radium of 5 pCi/L, it is likely that 25,000 to 30,000 would be not. Taking both the lower combined radium group and the group exceeding 15 total alpha into consideration, the upper limit of 21 radium-related cases may really be underestimated. It is instructive to compare the EPA estimate of risk with the data provided from the radium drinking water and bone cancer incidence risk from recent studies in Ontario, Canada (29). The Canadian study estimated a 17% increase in bone sarcoma risk from a daily ingestion increase of radium of about 30% above background (13 mBq/L in cases vs. 10 mBq/L in controls). This amounts to 1.7E 10-6 increased annual risk per 0.1 pCi/L increase in radium content of drinking water, or about 1.7 E 10-5 increased annual risk per pCi/L. The annual increased risk predicted from the Ontario study is similar to that noted by EPA for cancer morbidity. Because of the relatively wide confidence intervals expected for the Canadian study, the predictions would be consistent with the EPA values.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	The risks to an individual from radium exposure are expected to persist for decades, since radium is deposited, with low turnover rates, in bone. The severity of bone sarcoma is high.
Size of population(s) affected	It is roughly estimated that between 100,000 and 300,000 New Jersey residents are exposed to drinking water above the combined radium MCL. Additional people could be exposed to high total alpha levels, to lower combined radium levels, and to radium via food products. Agricultural and dairy radium activity data was not identified in New Jersey.

Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	Moderately high uncertainty. Actual tap water concentrations of radium need better assessment, since treatment with water softening greatly reduces radiation levels. In addition, information is needed on radium residues in New Jersey agricultural and dairy product.
Potential for significant future change in this risk estimate (H,M,L) and brief description	Medium. Unit risk estimates are likely to remain the same for the next decade, but population exposure estimates could change.
potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, !, =,/ where + is improvement)	0 The largest environmental exposure is to private well owners. In the absence of new initiatives targeted to this population, significant reductions in statewide exposure are unlikely.
Potential for catastrophic impacts (H,M,L) and brief description	Low.
Extent to which threat is currently regulated	Regulatory standards currently exist, with MCLs for combined radium and for gross alpha radiation in drinking water. There is a need to further consider 224Ra, which is not currently included in standards. Private well data is sparse, and therefore may not be fully assessed to determine compliance with regulatory standards.
Relative Contributions of Sources to Risk (H,M,L)	Moderate contribution of radium to overall risk of bone sarcoma is possible.
Primary anthropogenic sources	
Large business/industry	Not important factor
Small business industry	Not important factor
Transportation	Not important factor
Residential	Residential drinking water sources and household water softening make important impacts on population exposure to radium.
Agriculture	Could be relevant. Data on radium in New Jersey grown agricultural and dairy products is currently lacking.

	Sludges from publicly owned treatment works facilities in south Jersey are land applied for beneficial re-use. Some samples have been found to contain increased radium-226 and radium-228. While preliminary results of radium levels in the soil where sludges are applied are consistent with background, further study needs to be done.
Recreation	Radium content of fish and game could be important to some populations with high consumption.
Allocation of stressor-specific risk to primary NJ sources.	Vast majority of exposure and risk is due to natural sources of radium in drinking water. Only small contribution of agricultural fertilizer use.
Secondary anthropogenic sources	
Sediment sinks	Radium content of Ancient Hudson River basin is important.
Soil sinks	Radium content has been studied, varies across state with soil/rock/sand composition.
non-local air deposition	Not important radium source in NJ.
Groundwater sinks	Groundwater remains important source.
Surface water sinks	Radium not expected to be high in surface water.

Issue Summary: Radium

What is it?
Radium is a naturally occurring radioactive element that exists in rocks, soil, and groundwater. The main route of exposure to humans is via drinking water, although certain foods accumulate radium and may pose a significant source. There are also contaminated sites where historical use of radium has resulted in the potential for small populations to receive additional exposures.

What's at risk?
The risk varies with geographic region, mainly related to the level of radium in drinking water. The main risk to humans at exposures likely to be encountered in the New Jersey environment is cancer, including bone, lung, and stomach cancer. Drinking water sources with the potential for elevated radium levels appear to be confined to groundwater

Issue: Radium
Author: Jenny Goodman
Version: 01/28/00
sources.

What are the human health impacts in New Jersey?
In some areas of the state more than 50% of drinking water wells exceed health based standards. The total number of individuals with significant exposure depends not only on the particular source of the drinking water, but also on the extent and type of water treatment. It is estimated that statewide, 100,000 – 300,000 individuals use water which exceeds the drinking water standard. In many cases ground water provides only a portion of the drinking water supply with the remainder the result of mixing from surface water. Calculations of average exposure suggest that the risks from radium in drinking water can be expected to result in 21 additional lifetime cancers, which is less than one per year for the New Jersey population. However, there are significant uncertainties in these calculations and the actual numbers could be higher or lower. Individuals living near hazardous waste sites may be exposed to higher levels, but the additional population risk should be small.

What's being done?
There are regulations in place to monitor the levels of radium in drinking water from public water supplies. Exceedance of standards lead to action to reduce exposure. Private water supplies are not monitored or regulated.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe) BONE SARCOMA	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
Drinking Water H4	H 5	Y 4	M/H 4.3
NPL Site Work L1	L1	Y 3	L 1.7
			M/H 4.0

Drinking water is the major source of radium exposure for the general population of New Jersey, and is therefore more heavily weighted in the overall ranking than is the much smaller contribution of NPL sites. An estimate is that the MCL drinking water supply concentration is exceeded for both radium 226 and for total alpha for about 100,000 to 300,000 people in the state. Radium is a known cause of bone sarcoma, as documented in occupational health studies of radium dial painters, etc. A NOAEL for radium has not been found, and would be theoretically difficult to determine from human studies. Bone sarcoma increases have been reported in relationship to radium in drinking water in studies in Canada, Illinois, and Iowa. Because of these findings, and unknowns regarding actual tap water concentrations in many well water sites, elevated radium in some New Jersey drinking water sources is rated an important environmental health/ public health issue for the State.

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Issue: Radon
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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Radon**
Description of stressor (including etiology)	Radon is a radioactive gas. Radon-222 is a decay product of radium-226 which is found both naturally and technologically enhanced. Radon-222 is naturally occurring in outdoor air and groundwater and it can be found in concentrated amounts in indoor air.
Stressor-specific impacts considered including key impacts	Radon-222 - lung cancer from inhalation and stomach cancer from ingestion of drinking water (4).
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	Radon-222 - inhalation of indoor and outdoor air, ingestion of drinking water and inhalation from the release of radon gas from water use, showering, dishwashing, etc.
Population(s)/ecosystem(s) exposed statewide	Radon –222 in water: Public and Private well radon in water concentrations range from 9 pCi/L to 170,000 pCi/L. Radon-222 in air: Every person in the state is exposed to radon in both indoor and outdoor air.
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	Radon in indoor air: High. The average indoor radon concentration in New Jersey is 2.5 pCi/L. This level translates to a risk of 1.5×10^{-2}.(4,7) Radon in water: Moderately High. Needs further study, but initial data indicates Tier 1 and Tier 2 municipalities may have radon in water levels exceeding the proposed EPA's MCL of 300 pCi/L.
Specific population(s) at increased risk	Radon in air: Approximately 1,210,100 residents living in Tier 1 areas. Tier 1 is classified as a municipality having 25% or more homes tested with radon concentrations greater than 4 pCi/L (the EPA's current guideline). There are approximately 4,000,000 residents in Tier 2 areas. Tier 2 is classified as municipalities having between 5 and 24% homes tested with radon concentrations greater than 4 pCi/L. There are approximately 3,000,000 residents in Tier 3 areas. Tier 3 is classified as municipalities having less than 5% homes tested with radon concentrations greater than 4 pCi/L. Radon in water: Approximately 5 million

Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	Radon in air: The average indoor radon concentration in Tier 1 areas is 4.3 pCi/L. The average indoor radon concentration in Tier 2 areas is 1.9 pCi/L. The average indoor radon concentration in Tier 3 areas is 1.1 pCi/L. The average outdoor radon concentration is 0.4 pCi/L. Radon in water: The average radon in water concentration of affected communities is 2,100 pCi/L. (9)
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	Current studies suggest that radiation exhibits a linear, no threshold dose-effect relationship (6). Lifetime Unit Risk Radon in Air – $5.9 \times 10^{-3}(pCi/L^{-1})$ Radon in Water – $6.7 \times 10^{-7}(pCi/L^{-1})$
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	Radon in indoor air: Tier 1- Cancer risk at average concentration equals 2.5×10^{-2} (4,7). Expected number of lung cancers/year is 430. Tier 2- Cancer risk at average concentration equals 1.1×10^{-2} (4,7). Expected number of lung cancers/year is 630. Tier 3- Cancer risk at average concentration equals 6.5×10^{-3}. (4,7) Expected number of lung cancers/year is 280. Outdoor air- Cancer risk at average concentration equals 2.3×10^{-3}. (4,7) Expected number of lung cancers/year is 260. Radon in water: Cancer risk at average concentration equals 1.35×10^{-3}. (7) (This includes the ingestion and inhalation risks.) Expected number of lung cancers/year is 86 and stomach cancers/year is 10. Total expected number of lung cancers/year for NJ is 1426. Total expected number of stomach cancers/year in NJ is 10.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Lung cancer should be considered severe and largely irreversible. Radon in air: Effects are from chronic exposure. Assumption is a lifetime exposure of 70 yrs and inhalation rate of $16.8 m^3/d$. (4) Radon in water: Assumption is a lifetime exposure of 70 yrs and ingestion rate of 2 L/d. (7)
Size of population(s) affected	Radon in air: Entire State, approximately 8 million. Radon in water: Approximately 5 million. All NJ citizens are at high risk from radon ($>10^{-4}$). The entire state population is exposed to radon in the outdoor air which has an associated risk of 2.3 x 10 –3.

Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	L: There is not a significant uncertainty in the exposure estimates for NJ residents. The Radon Program within the NJDEP has a large radon testing database with the results of over 400,000 tests that were conducted in NJ homes. This data was used to establish three radon potential areas or Tiers within NJ. The risk computations are based on 11 cohorts of underground miners. The absorbed dose delivered to the target cells in the bronchial epithelium differs somewhat among mines and among different homes. The various factors such as higher unattached fraction in homes versus mines and lower breathing rates in homes versus mines compensate, in such a way that the application of the models for exposure in homes derived directly from the mines is considered valid for predictive purposes. (7) Case-control epidemiological studies have supported the risk of radon in air(4, 8).
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	L: The National Academy of Science is convening the Biological Effects of Ionizing Radiation Committee to address the dose-response curve. This report is not expected for several years.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , ~, where + is improvement)	Radon in air: ++ With continued outreach program, more residents will test their homes and mitigate if necessary. Effective mitigation systems can reduce radon exposures by over 80%. Radon in water: 0 Effective radon removal systems include aeration, however, currently there is no requirement for private home owners to test their wells for radon in water. Public water supplies will be required to test when the EPA finalizes its radon in water rule.
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	Radon in indoor air: L Radon in drinking water: L
Extent to which risks are currently reduced through in-place regulations and controls	Radon in air: There are no requirements that a homeowner must test their home for radon, however, it has become standard practice in real estate transactions. Daycare centers are required to test for radon through DYFS regulations. The Dept. of Community Affairs regulates new construction via the Radon Hazard Subcode ((N.J.A.C. 5:23-10). Newly constructed houses in Tier 1 areas must incorporate radon-resistant construction techniques. Radon in water: The EPA is expected to promulgate a radon in water MCL in the near future which incorporates a multi-media mitigation approach. This means that water companies may opt to treat homeowner's radon in air rather than treat the drinking water for radon.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	Radon in indoor air: L Radon in water: L
Small business industry	Radon in air: M When public water companies are required to mitigate radon in water, the aeration treatment will increase the outdoor radon levels close to the treatment plant. This will probably require an air permit. Radon in water: L

Transportation	Radon in indoor air: L Radon in water: L
Residential	Radon in indoor air: L Radon in water: L
Agriculture	Radon in indoor air: L Radon in water: L
Recreation	Radon in indoor air: L Radon in water: L
Resource extraction	Radon in indoor air: M Radon in water: L
Government	Radon in indoor air: L Radon in water: L
Natural sources	Radon in indoor air: H Radon in water: H
Contaminated sites	Radon in indoor air: M Radon in water: M There are a number of contaminated sites (10) in NJ that are contaminated with technologically enhanced naturally-occurring radioactive materials. In some cases, his material was used as fill during residential construction activities. New Jersey citizens were living in homes built on radium contaminated soil and consequently, extremely high levels of radon were found in their homes. There may be yet undiscovered sites where this is the case as well.
Diffuse and non-NJ sources	
Sediment	Radon in indoor air: N/A Radon in water: N/A

Soil	Radon in indoor air: N/A Radon in water: N/A
Non-local air sources (including deposition)	Radon in indoor air: L Radon in water: L
Biota sinks	Radon in indoor air: L Radon in water: L

Human Health Issue Summary: Radon

What is it?
Radon is a radioactive gas that is emitted during the decay of uranium, a naturally occurring mineral found in New Jersey rocks and soil. While radon gas is not a threat in the ambient (outdoor) air, it can become concentrated in buildings where it enters and collects in basements. At these concentrated levels, radon is a human carcinogen. When radon is inhaled, small radioactive particles are retained in the lungs, increasing the risk of lung cancer. Radon may also be present in drinking water, and exposure via ingestion of contaminated water increases the risk of stomach cancer.

Who's at risk?
Some individuals are exposed to greater concentrations of radon because of the location and/or construction of their homes or businesses. Houses and other structures contain varying concentrations of radon gas due to differences in the radon content of underlying soils and rocks, and because of differences in ventilation. Smokers are at an increased risk because the rate of cancer growth in smokers is increased with exposure to radon.

What is the extent of human health problem in New Jersey?
The total number of lung cancers resulting from radon exposure may be as high as 1700 per year. The number of stomach cancers attributable to radon may total 10 per year.

What's being done?
Legislation has been proposed for new home construction and mitigation of drinking water risks in areas with elevated radon levels. New Jersey citizens are encouraged to monitor their homes for radon.

Issue: Radon
Author: Jenny Goodman
Version: 01/11/01
Reviewer Ranking of Stressor

Date 12/29/00

Stressor Radon-222

Reviewer Jenny Goodman

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population affected integrating across health effect) (H.M.L) (also 1-5 with 1 being the lowest overall risk)
H	H	Y	H
5	5	5	5

References:

1. Radon, Radium and Uranium in Drinking Water, C. Richard Cothern and Paul A. Rebers, 1990.

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3. EPA Federal Guidance Report No. 13, Cancer Risk Coefficients for Environmental Exposure to Radionuclides, EPA 402-R-99-001, September, 1999.

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5. National Council on Radiation Protection and Measurements Report No. 94, Exposure of the Population in the United States and Canada from Natural Background Radiation, 1987.

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8. “Residential Radon Gas Exposure and Lung Cancer”, American Journal of Epidemiology, Vol. 151, No. 11, June, 2000.

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Issue: Secondhand Tobacco Smoke
Author: Bilue Thomas
Version: 08/00

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Secondhand Tobacco Smoke (STS)**
description of stressor (including etiology)	STS is a complex mix of chemicals generated during the burning and smoking of tobacco products. STS includes the emission from the burning end of the cigarette, cigar, or pipe as well as the exhaled mainstream smoke. All the compounds found in mainstream smoke, the smoke inhaled by the active smoker, are also found in "side stream" or secondhand tobacco smoke. Over 4,000 chemicals including 40 known or suspected human carcinogens have been identified in cigarette smoke. Chemicals present in STS include irritants and systemic toxicants such as hydrogen cyanide and sulfur dioxide; mutagens and carcinogens such as benzo (a) pyrene, formaldehyde, 4-aminobiphenyl; and the reproductive toxicants, nicotine, cadmium, and carbon monoxide[1].
stressor-specific impacts considered including key impacts Lung Acute	The major impacts of STS exposure are related to morbidity as the following list clearly indicates. The effects are ranked below in order of number of cases reported annually at the national level[2]: Otitis media (middle ear infections) Asthma exacerbation Bronchitis and Pneumonia New Asthma cases Ischemic heart disease Low Birth weight Cancer Sudden Infant Death Syndrome (SIDS) Lower R Respiratory Tract Illness (LRI) in children up to 18 months
Exposure Assessment	
exposure routes and pathways considered (include indoor air as appropriate)	In New Jersey, exposure to STS occurs primarily in the residential and recreational environments. It occurs to a lesser degree in the work environment due to regulations on smoking in the workplace. Exposure to the carcinogens in tobacco smoke occurs when side stream smoke is inhaled by the non-smoker into the lungs and breathing passageways of the body. It is then distributed to various sites in the body thereby contributing to carcinogenesis.
population(s)/ecosystem(s) exposed statewide	STS exposure occurs among all populations and ecosystems throughout New Jersey. The 1997 Behavioral Risk Factor

	Survey revealed that 21.3% of New Jersey adults, aged 18 and older, were current cigarette smokers. The 1996 data also reveal that the age group 18-24 had the highest percentage of usage with 29.6% reporting a positive smoking status. And the most recent New Jersey Youth Tobacco Survey (1999) found that 14.6% of middle school students and 28.9% of high school students are current smokers. Additionally, the results of the 1999 Behavioral Risk Factor Survey reveal that 25% of smokers have reported smoking indoors in the past 30 days*. * These figures are based on unweighted raw data obtained from the 1999 survey-official results may differ from those reported here.
quantification of exposure levels statewide (include indoor air as separate category as appropriate)	Quantification of exposure at the state level in New Jersey is done through the use of surveys and questionnaires. New Jersey state has compiled data on tobacco use through the use of statewide reports, such as the 1999 New Jersey Youth Tobacco Survey and through participation in the CDC's Behavioral Risk Factor Surveillance System.
specific population(s) at increased risk	Children's lungs are even more susceptible to harmful effects from STS than those of adults. In infants and young children up to three years old, exposure to STS causes an approximate doubling in the incidence of pneumonia, bronchitis, and bronchiolitis. There is also strong evidence of increased middle ear effusions, reduced lung function, and reduced lung growth. Several recent studies link STS with increased incidence and prevalence of asthma and increased severity of asthma symptoms in children of mothers who smoke heavily. These respiratory illnesses in childhood may contribute to small, but significant lung function reductions associated with exposure to STS in adults[3].
quantification of exposure levels to population(s) at increased risk (due to factors other than exposure) (include indoor air as separate category as appropriate)	Quantification of exposure levels to children, neonates or asthmatics has not occurred at the state level.
Dose/Impact-Response Assessment	
quantitative dose/impact-assessment employed for each population considered	According to USEPA's carcinogen classification system, STS is considered a □Group A□ carcinogen. The dose-response relationship is such that effects are observable at low doses and there is no evidence of a threshold in any population considered.
Risk Characterization	
risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	The population of the state of New Jersey accounts for roughly 2% of the US population. Extrapolating from the number of cases annually, it is estimated that the following number of cases/deaths occur in New Jersey annually: Otitis media:14,000-32,000 cases Asthma exacerbation: 8,000-20,000 cases

	Bronchitis and Pneumonia: 3,000-6,000 cases New Asthma Cases: 160-520 cases Ischemic Heart Disease: 700-1,240 deaths Low Birth weight: 194-372 cases Lung Cancer: 60-80 deaths annually Sudden Infant Death Syndrome: 38-54 deaths Acute LRI (Lower Respiratory Tract Infections)*: 2-4 deaths *Numbers are based on figures in Health Effects of Exposure to Environmental Tobacco, The Report of the California Environmental Protection Agency - see reference for full citation.
assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	The health effects of exposure to STS vary widely. The overwhelming number of reportable effects are associated with reversible conditions and morbidity as a result of STS exposure is more widespread than mortality. Conditions associated with STS exposure include: <u>In adults:</u> Rhinitis/pharyngitis, nasal congestion, persistent cough Conjunctival irritation Headache Wheezing Exacerbation of chronic respiratory conditions In children and infants: Asthma onset Increased severity of or difficulty in controlling asthma Frequent upper respiratory infections and/or episodes of otitis media Persistent middle ear effusions Noring Repeated pneumonia, bronchitis
	Over half, (53%) of *all* effects (including lung cancer and death due to ischemic heart disease) manifests as middle ear infections, occurring mostly in children. Asthma exacerbation accounts for 33% of all affects. Bronchitis and Pneumonia, also treatable, account for 10% of STS related illness.

	Ischemic Heart disease which usually ends in death, accounts for the majority of morbidity associated with STS exposure, followed by deaths due to lung cancer.* *Percentages are based on figures in Health Effects of Exposure to Environmental Tobacco, The Report of the California Environmental Protection Agency - see reference for full citation.
size of population(s) affected	As noted above estimates for New Jersey include: Otitis media: 14,000-32,000 cases Asthma exacerbation: 8,000-20,000 cases Bronchitis and Pneumonia: 3,000-6,000 cases New Asthma Cases: 160-520 cases Ischemic Heart Disease: 700-1,240 deaths Low Birth weight: 194-372 cases Lung Cancer: 60-80 deaths annually Sudden Infant Death Syndrome: 38-54 deaths Acute LRI (Lower Respir. Tract Infections): 2-4 deaths
assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	Exposure to STS has generally been determined in three ways: ascertainment of spousal smoking status; estimation of the number of hours a person is exposed at home, at work, or elsewhere or measurement of biomarkers. Characterization of STS exposure in most epidemiological studies is limited to broad categories (e.g., yes/no, number of hours per week). Accurate categorization is difficult given the large variation in exposures individuals experience due to length of exposure, individual variability in metabolizing carcinogens, etc.
potential for significant future change in this risk estimate (H,M,L) and brief description	Low to Medium Risk estimates may change if exposure can be more accurately recorded, or if the mechanisms between exposure and disease are better understood. But as this would require the development of new laboratory techniques or more vigilant surveys on smoking status, the likelihood of any change in risk estimates in the near future is low to medium.
potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	N/A
extent to which risks are currently reduced through in-place regulations and controls	In the 1970's, New Jersey was a leader in restricting the non-smoker's exposure to STS. Current state laws only require that restaurants declare themselves to have a non-smoking section; it does not require a non-smoking section, nor does it require a separate ventilation system even if there is a non-smoking section. Current state laws also do not prohibit smoking in the workplace. Rather, it requires those employers with more than 50 employees have a smoking policy. It does not require any separation between smoking and non-smoking areas and although smoking in public places is prohibited, State laws do permit smoking areas under certain conditions. However, the results of the 1999 Behavioral Risk Factor Surveillance System show that 80.6% of respondents have a

	policy of no smoking in designated work areas. Only 10% report some areas in which smoking is permitted and 6% with no official policy, while 2% have smoking in all work areas*. Most of the more recent restrictions on exposure to STS have occurred at the municipal level. Some have a complete ban on smoking, while others have banned smoking in public places that are frequented by children; others have bans on smoking in restaurants and workplaces[4]. Furthermore, commercial day care centers are required to be smoke-free. * These figures are based on unweighted raw data obtained from the 1999 survey-official results may differ from those reported here.
Relative Contributions of Sources to Risk (H,M,L)	The following sources are categorized as High and Low[5] High contributors: residences, bars, shopping malls, enclosed arenas, hotels and motels because they have no restrictions on smoking. Low-Medium contributors: government and private work sites and commercial daycare centers.
Allocation of stressor-specific risk to primary NJ sources	
large business/industry	N/A
small business industry	N/A
transportation	N/A
residential	High
agriculture	N/A
recreation	High
resource extraction	N/A
government	N/A
natural sources	N/A

contaminated sites	N/A
diffuse and non-NJ sources	
sediment	N/A
soil	N/A
non-local air sources (including deposition)	N/A
biota sinks	N/A

Issue: Secondhand Tobacco Smoke
Author: Bilue Thomas
Version: 08/00

Human Health Issue Summary: Secondhand Tobacco Smoke

What is it?
Secondhand Tobacco Smoke (STS) is a complex mix of chemical generated during the burning and smoking of tobacco products. It is also known as passive smoke. Over 4,000 chemicals, including 40 known or suspected carcinogens have been identified in cigarette smoke. Exposure to STS can cause or contribute to middle ear infections, asthma, bronchitis and pneumonia, ischemic heart disease, low birth weight, lung cancer, Sudden Infant Death Syndrome (SIDS), and Acute Lower Respiratory Tract Illness (LRI) in children up to eighteen months. All of the compounds found in the smoke inhaled by the active smoker are also found in secondhand smoke.

What's at risk?
Children are more susceptible to the harmful effects of STS than adults, although all persons breathing in secondhand smoke are at risk. In infants and young children up to three years old, exposure to STS causes an approximate doubling in the incidence of pneumonia, bronchitis, and bronchiolitis. There is also strong evidence of increased middle ear infection, reduced lung function, and reduced lung growth.

What are the human health impacts in New Jersey?
STS is considered a 'Group A' cancer-causing substance, meaning that the health effects are noticeable at low doses, and there is no evidence that any particular group of individuals will remain unaffected. It is estimated that the following number of cases/deaths occur in New Jersey annually:

- Middle Ear Infection, 14,000-32,000 cases
- Asthmatic episodes, 8,000-20,000 cases
- Bronchitis and Pneumonia, 3,000-6,000 cases
- New Asthma Cases, 160-520 cases
- Ischemic Heart Disease, 700-1,240 deaths
- Low Birth Weight, 194-372 cases
- Lung Cancer, 60-80 deaths
- Sudden Infant Death Syndrome, 38-54 deaths
- Acute Lower Respiratory Tract Infection, 2-4 deaths

In New Jersey, 53% of all effects (including lung cancer and death due to heart disease) manifest as middle ear infections, occurring mostly in children. Asthma exacerbation accounts for 33% of all effects. Ischemic heart disease, which usually ends in death, accounts for the majority of deaths associated with STS exposure, followed by deaths due to lung cancer.

What's being done? Most restrictions on exposure to STS have occurred at the municipal level, where restaurants, and public places may have smoking bans. Commercial daycares are required to be smoke-free. State regulations do not restrict smoking in bars, shopping malls, hotels, or enclosed arenas.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L)

		there are discrete communities at elevated risk)	(also 1-5 with 1 being the lowest overall risk)
5 - H	**4 - H**	**N**	**5 -H**
			5 -H

[1] NIH, NCI, Cal EPA Study

[2] NIH, NCI, Cal EPA Study

[3] USEPA, Indoor air pollution: A Guide for Health Professionals.

[4] New Jersey Department of Health and Senior Services: Strategic Plan for a Comprehensive Tobacco Control Program.

[5] MMWR, June 25 1999 Summary of State Laws by type of Restriction and state as of December 31,1998.

Issue: Secondhand Tobacco Smoke
Author: Bilue Thomas
Version: 08/00
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Issue: Sulfur oxides (SOx)/Sulfates
Author: Ken Jones, Michele Witten
Version:04/20/01

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	**Sulfur oxides (SOx)/Sulfates**
Stressor	
Description of stressor (including etiology)	Oxides of Sulfur are by-products of combustion of sulfur containing fuels. The dominant fuel producing SO_x is coal, as coal has significant concentrations of sulfur. Liquid petroleum fuels have less sulfur, and are therefore less significant as sources of SO_x. In the atmosphere, SO_x can be converted to sulfuric or sulfurous acid and result in acid precipitation. The ecological impacts from acid precipitation are covered in a separate analysis.
Stressor-specific impacts considered including key impacts	As with most air pollutants, the primary impacts from SO_x are to the respiratory tract. Reduced lung capacity and irritation of the bronchial tract are potential impacts related to SO_x inhalation. The secondary results of these effects can be increased respiratory disease and a reduced capacity to respond to infection.
Exposure Assessment	
Exposure routes and pathways considered **(include indoor air as appropriate)**	SO_x primary route of exposure is via inhalation. Often SO_x is associated with particulates, because SO_x adheres to particulates, and combustion processes result in particulate generation as well as SO_x generation. The consequence of this association complicates laboratory assessments because particle associated SO_X behaves differently than gaseous SO_x. Epidemiologic studies incorporate this synergistic effect because of the common appearance of SO_x and particulates.
Population(s)/ecosystem(s) exposed statewide	For this analysis, we consider two populations. There is an average exposure that all New Jersey citizens face, and there are some areas in the state that face elevated concentrations. We identify four monitors that have shown hourly readings greater than .05 ppm during 2000 (EPA, (IRIS)). These four counties are Camden, Gloucester, Morris and Union. Because Essex County does not have a monitor, we make the cautious assumption that Essex County may also face elevated readings as have the neighboring counties of Morris and Union.
Quantification of exposure levels statewide, including populations at significantly increased exposure **(include indoor air as separate category as appropriate)**	SO_x is measured at 13 monitoring stations across the state covering 10 counties. In the high risk areas represented by the four monitors described above, the average concentrations over the year are still well below national standards at between .0044 and .0085 ppm. Furthermore, none of the sites have readings above .05 ppm more than 1% of the time. (EPA, (IRIS)) For the rest of New Jersey, the worst 10% of SO_x readings are typically below .015 ppm.

category as appropriate)	
Specific population(s) at increased risk	• Children are at risk for the reduced capacity to respond to infection. • Asthmatics show lung function responses to SO_x exposure.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations)	In the five counties with elevated SO_x, there may be some reduction of a child's capacity to fight infection, although the low frequency of these exposures may suggest minimal incidence of these effects. The possibility for increased cases of respiratory disease suggested for concentrations between .01 and .05 is enhanced throughout the state, however the evidence drawing this conclusion is less certain than other epidemiological studies (NAPAP).
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	The impacts from SO_x are difficult to separate from impacts of associated pollutants. In particular, there has been a significant correlation between elevated SO_x and particulates, especially in those cases where extreme pollution events led to easily observed impacts (such as the deadly events in London during the 1950s). Clinical studies for SO_x focus upon exposures greater than 1 ppm, which are no longer relevant to most environmental exposures. Therefore, the impacts described in this assessment are the result of epidemiological studies at concentrations between .01 ppm and 1 ppm (NAPAP). • At concentrations above 0.2 ppm increased asthmatic reactions are apparent. • At concentrations between .05 and .2 ppm there is significant evidence of a reduced capacity to respond to infection. This is noted by both increased incidence of infection and changes in immune system function (Lippmann, 1991). • At concentrations between .01 and .05 ppm there is some evidence of increased respiratory illness in children (NAPAP).
Risk Characterization Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	There are two standards established under the Clean Air Act in the U.S.; a 24 hour average (.14 ppm), and an annual average (.03 ppm). In the five counties with incidences of elevated SO_x, the population of children age 0-3 is 100,000 and the asthmatic population is 75,000 (assuming a 3% asthma rate) (American Lung Association). Therefore, we can assume that the children in the urban counties have a slight but increased chance for both respiratory illness and for greater susceptibility to colds and flu symptoms. Throughout the state, there is a small chance that all children will have an increased frequency of respiratory disease. We can also assume that there is a remote chance that asthmatics will be affected by levels of SOx that exist in the counties with greatest readings.
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Respiratory disease is familiar to most of us who have ever suffered through a bad cold with periods of coughing. The reduced ability to combat infection can cause a range of effects from increased inflammation, due to infection, to accelerated death in those with already compromised immune systems.
Size of population(s) affected	The total population of New Jersey children that may have an increased rate of respiratory disease is 330,000. The population of children living in areas of high concentration that may suffer a reduced capacity to respond to infection is

	100,000.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	M-L As we have noted above, the principle uncertainty in assessing the damage from SO_x is the complicating factor of particulates as a pollutant that is often present together with SO_x.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	L – In the recent past, we have made significant reductions in SO_x concentrations (NJ DEP, 1999), while there are still many areas of the state and country with significant particulate pollution. Therefore, it is possible that new knowledge may arise identifying specific particulate impacts that will change some of the estimates from SO_x damage.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =, where + is improvement)	++ SO_x emissions are decreasing from most sources throughout the country (US EPA). The next set of potential reductions may occur if older power plants, grandfathered from regulations during early versions of the Clean Air Act, are the focus of further regulation.
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L – SO_x emissions are not likely to result from catastrophic events.
Extent to which risks are currently reduced through in-place regulations and controls	SO_x emissions have been reduced significantly as the result of implementation of Clean Air Act legislation. In New Jersey, the worst readings from monitoring sites thirty years ago are almost three times the levels seen today (NJ DEP, 1999). This trend is evident in other parts of the country as the result of significant point source regulation.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	M/H - The primary source of SO_x emissions remains large utility boilers. According to Environmental Defense, boilers account for almost 85% of the total point source emissions of SO_x, with the largest single source, a power plant in Burlington County releasing approximately 15 percent of the state total point source emissions. (Environmental Defense)
Small business industry	L
Transportation	L/M
Residential	L

Agriculture	L
Recreation	L
Resource extraction	L
Government	L
Natural sources	L
Contaminated sites	L
Diffuse and non-NJ sources	
Sediment	N/A
Soil	N/A
Non-local air sources (including deposition)	M A significant source of New Jersey ambient concentrations of SO_x remains from out-of-state sources.
Biota sinks	

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	**Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)**	**Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)**	**Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)**
Asthmatics 2-L/M	4-M/H	2-L/M	2-L/M
Children with increased respiratory disease 3-M	3-M	3-M	2-L/M
Children with decreased resistance to infection 3-M	2-L/M	3-M	2-L/M
			2-L/M

Issue: Sulfur oxides (SOx)/Sulfates
Author: Ken Jones, Michele Witten
Version:04/20/01

Issue Summary: Sulfur Dioxide (SO_2 /SO_x)

What is it?
Sulfur dioxide is the primary component of the class of air pollutants known as oxides of sulfur (SO_x). It is a product of fossil fuel combustion (primarily coal) and is a byproduct of several chemical processes such as paper manufacture and smelting. Together with oxides of nitrogen, SO_x is a component of acid precipitation, the impacts of which are discussed separately. Elevated concentrations of SO_x can result in respiratory effects such as reduced lung capacity and irritation to the bronchial tract, which may lead to respiratory disease and a reduced capacity to respond to infection.

What's at risk?
The general population is exposed to low levels of SO_x statewide. At particular risk are asthmatics, for whom exposure to SO_x increases incidence of asthma attacks. Children are also at risk of an increased incidence of respiratory disease, and there is some evidence that SO_x exposures reduces their ability to respond to infection.

What are the human health impacts in New Jersey?
SO_x levels in New Jersey are well below federal health-based standards. Concentrations in Camden, Gloucester, Morris, and Union counties were slightly elevated relative to other areas in 2000. There are no monitors in Essex County and slightly elevated concentrations of SO_x may exist there as well. There is evidence that even these relatively low levels may result in a reduced capacity to respond to infection in some individuals, placing about 100,000 children in these five counties potentially at risk. There is also some evidence that SO_x results in increased incidence of respiratory disease in children.

What's being done?
Federal regulations have reduced the emissions from most point sources significantly and the highest levels recorded in New Jersey today are about one third of what they were 30 years ago. Since 1995, EPA has operated a market-based allowance trading system, which provides economic incentives for regulated facilities to reduce their emissions below required levels.

References:

American Lung Association, *Trends in Asthma Morbidity and Mortality*, http://www.lungusa.org/data/asthma/asthmach_1.html#introduction, January, 2001.

Environmental Defense, *Scorecard*, www.scorecard.org, April, 2001.

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National Acid Precipitation Assessment Program (NAPAP), 1990, *Direct Health Effects of Air Pollutants Associated with Acidic Precursor Emissions*, SOS/T, Vol. 22.

New Jersey Department of Environmental Protection, 1999, *Air Goal Environmental Progress Briefing.*

US Environmental Protection Agency, Aerometric Information Retrieval System (AIRS), www.epa.gov/airs/airs.html

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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Ultraviolet (UV) Radiation**
Description of stressor (including etiology)	Naturally occurring UV Radiation is an electromagnetic wave produced by the sun. It has been divided (somewhat arbitrarily) into three categories: UVA, with wavelengths from 320-400; UVB, with wavelengths from 280-320 nanometers[1]; and UVC, with wavelengths from 100-280 nanometers. UVA is not absorbed by the ozone layer, but is considered less hazardous than UVB, which is mostly absorbed by the ozone layer. UVC is extremely hazardous, but at the present time it is completely absorbed by atmospheric ozone and normal oxygen (O^2). While atmospheric ozone is in and of itself not a stressor, it is recognized as a primary filter through which UV must pass to reach the surface of the earth. Degradation of this filter therefore should be considered when assessing UV as a hazard and shall be included in this assessment. Of note is the news release by the National Oceanic and Atmospheric Administration (NOAA) where researchers reported that the September 2000 decline in ozone occurred about six days earlier than in any previous year. In addition, the ozone hole peaked at 28.5 million square kilometers and is the largest geographical size on record. The ozone hole is defined as the size of the region with total ozone below 220 Dobson units. (NOAA) Most artificial sources of UV, except for lasers, emit a spectral continuum of UV containing characteristic peaks, troughs and lines. These sources include various lamps used in medicine, industry, commerce, research and the home.[2]
Stressor-specific impacts considered including key impacts	UV exposure is known to be associated with various skin cancers, accelerated skin aging, cataract and other eye diseases, and may reduce a person's ability to resist infectious diseases. The U.S. Department of Health and Human Services included in its "Ninth Report on Carcinogens" that both solar radiation and exposure to sunlamps or sunbeds are listed as "known to be a human carcinogen". Thinning of the ozone layer and "holes" in this layer will contribute to increased levels of all UV associated diseases, especially to unprotected and/or at risk populations. While artificial exposure to UVA has been widely used for cosmetic reasons and is often considered "safe" by the general public, a recent study published in the British Journal of Cancer provides evidence for UVA carcinogenicity and that exposure to sunbeds might increase the risk of developing malignant melanoma.[3]
Exposure Assessment Exposure routes and pathways considered (include indoor air as appropriate)	The primary source of UV radiation for the majority of humans is exposure to the Sun. 50% of UV emitted by the sun which reaches the Earth occurs between 11:00am and 2:00pm, so this is a critical window of exposure. Additional factors which influence an individual's exposure to natural UV include: latitude and elevation, cloud cover, proximity to an industrial area, and albedo (reflection).
	Artificial sources of UV abound, but the greatest exposure to the general population comes from the use of sunbeds. These devices

<table>
<tr><td></td><td>are intended to produce a tan by emitting UVA and some UVB.
Additional artificial UV radiation occurs from UV sources used for a variety of diagnostic and therapeutic medical purposes, industrial and commercial processes, and fluorescent lamps, (which emit small amounts of UV).
Tungsten halogen lamps are used in the home and in the workplace for a variety of lighting and display purposes. Unshielded lamps can emit UV levels sufficient to cause acute injury at short distances.
Arc welding produces very high levels of UV. Eye and skin protection is mandatory for workers, but caution must be taken to insure that accidental exposures to unprotected individuals in a work area does not occur.</td></tr>
<tr><td>Population(s)/ecosystem(s) exposed statewide</td><td>The entire population of NJ (with unusual exceptions) will have some exposure to naturally occurring UV during the course of a normal day. Because of the numerous opportunities for outside recreation in the State, NJ also hosts a large summertime population of out of state residents who flock to our coastal areas, primarily during the summer months.
All populations are susceptible to eye damage from exposure to UV, however susceptibility to skin cancers and UV burn typically affect lighter skinned individuals to a greater extent then darker skinned individuals. Exposure levels are determined by a number of variables such as duration of exposure, skin type and UV intensity during the time of exposure.

Determining UV Radiation exposure depends on three primary factors:

Amount of UV radiation at a measured location (NJ) in question;
Time of day and duration of exposure; and
Physical characteristics and personal behaviors which can determine the degree to which UV is absorbed by the body.</td></tr>
<tr><td>Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)</td><td>Measuring UV is relatively easy with the proper instrumentation. It is more difficult however to measure individual exposure because of the number of variables. The amount of solar radiation at any given location varies by day and time of day because of the changing relation to the sun. Factors which influence an individual's exposure to natural UV include: latitude and elevation, cloud cover, proximity to an industrial area, and albedo (reflection). Each of these factors makes quantification of exposure difficult. We can however quantify exposure risk using the UV Index. The Index predicts UV intensity levels on a scale of 0 to 10+, where 0 indicates a minimal risk of overexposure and 10+ means a very high risk. Calculated on a next-day basis for dozens of cities across the United States, the UV Index takes into account clouds and other local conditions that affect the amount of UV radiation reaching the ground in different parts of the country. (USEPA) The table below provides a guide to the index. Exposures in NJ for June of 2000 were typically in the 7 to 8 range, or in other words, high risk.

<table>
<tr><th>Number</th><th>Exposure Level</th></tr>
<tr><td>0 to 2</td><td>Minimal</td></tr>
<tr><td>3 to 4</td><td>Low</td></tr>
<tr><td>5 to 6</td><td>Moderate</td></tr>
<tr><td>7 to 9</td><td>High</td></tr>
<tr><td>10+</td><td>Very High</td></tr>
</table>
</td></tr>
<tr><td></td><td>So, the UV index provides some quantification of exposure RISK on any given day in New Jersey, but an assessment of the UV</td></tr>
</table>

	exposure of a population remains more difficult to quantify. Time of day and duration of exposure are variables which are difficult to track in any meaningful way for a population. The time of day an individual is exposed is vital to determining exposure levels. 50% of UV emitted by the sun which reaches the earth occurs between 11:00am and 2:00pm and so this is a critical window of exposure. The duration a person is exposed during the day is likewise important in determining total exposure. An individual's physical characteristics (primarily skin pigment) and personal behaviors (the degree to which they will protect themselves from exposure using sunscreens, sunglasses, etc.) determine the degree to which UV is absorbed by the body. While it is true that light skinned individuals are more likely to experience UV related damage to their skin, all populations are potentially affected by UV exposure to the eye and possible effects on the immune system.[9] Given the difficulties as noted above for determining exposure levels, we must focus instead on exposure risk, which is easier to quantify and track, in attempting to communicate UV as a hazard.
Specific population(s) at increased risk	Lightly pigmented individuals to whom UV exposure causes sunburn but little tanning (e.g.,. Celtic populations) are typically at greatest risk. Characteristics of this group include fair or red hair, blue eyes and freckles. People in this group must take extra care in the sun as their skin is poorly protected and easily damaged. A small percentage of people have a skin condition that makes them particularly sensitive to the sun's UV rays; this is called photosensitivity. Photosensitivity disease (porphyria) and photo-aggravated disease (e.g. lupus erythematosus) are triggered by minimal UV exposures. In addition, some medications, foods and cosmetics contain ingredients that may cause photosensitivity. This combination of chemicals or drugs with UV causes an adverse effect in the skin such as a rash or exaggerated sunburn. (World Health Organization: WHO/EHG/95.17) A segment of the population which frequents artificial tanning facilities are also at an increased risk. These individuals intentionally expose themselves to hazardous UV in order to achieve a temporary change in skin pigment.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	To properly determine exposure levels for those populations at increased risk of melanomas of the skin, quantification of the population of NJ by skin type would be necessary. Broad generalizations of skin type using categories such as "White", "Hispanic" and "Black" are not sufficiently detailed to provide meaningful risk assessment on State populations. Individual assessments are, in the absence of a detailed state survey of skin types, the only accurate way of determining risk. Any attempt at using the broad skin categories mentioned above would result in an overly broad risk assessment of the general population. In addition to the difficulties with statewide assessments using broad generalizations of skin type, exposure varies widely depending on the occupational and non-occupational activities of the target population. While a statewide study could be conducted to take into account all of the factors outlined above, to the knowledge of the author no such study has been conducted.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered .	As would be expected, hazards associated with UV exposure increase as the dose increases. Damage to the eyes and immune system affect all populations, while risk of melanomas of the skin affect individuals with sensitive skin types to a much greater degree than people with skin less affected by UV. While this seems rather general, the impact of UV radiation on the population will vary depending on factors previously outlined.

Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	Exposure is less in darker skin groups than light skin groups living in the same geographical area. Risk of skin cancer decreases with increasing pigmentation. Incidence of both melanoma and non-melanocytic skin cancer are increased in areas of high ambient solar UV radiation. (World Health Organization: WHO/EHG/95.16)
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	SKIN Chronic skin changes due to UV consist of skin cancer (both melanoma and non-melanocytic), benign abnormalities of melanocytes (freckles, melanocytic naevi and solar or senile lentigines), and a range of other chronic injuries resulting from UV exposure to keratinocytes, blood vessels and fibrous tissue, often described as "photoaging" (solar elastosis). The much increased rates of skin cancer in patients with xeroderma pigmentosum, who have a deficiency in the capacity to repair UV-induced DNA damage, suggest that direct UV damage of the DNA may be a step in the cause of these cancers. This suggestion has also been supported by the observation of UV specific mutations of the p53 tumor suppressor gene in a proportion of patients with non-melanocytic skin cancer. Oxidative and immune suppressant effects may also contribute to the capacity of UV to cause skin cancers. EYE The acute effects of UV on the eyes consist of the development of photokeratitis and photoconjunctivitis, which are unpleasant but usually reversible and easily prevented by wearing appropriate eyewear. Chronic effects on the eye consist of the development of pterygium and squamous cell cancer of the conjunctiva and cataracts. A review of the studies suggests that there is sufficient evidence to link acute ocular exposure to photokeratitis but knowledge of the effects of chronic exposure is less certain. While there is sufficient evidence that cortical and posterior subcapsular cataracts (PSC) can be caused by UVB in laboratory animals, there is limited evidence to link cortical and PSC cataracts in humans to chronic ocular exposure to UVB. IMMUNE SYSTEM A number of studies suggest that UV exposures at environmental levels suppress immune responses in both rodents and man. In rodents this immune suppression results in enhanced susceptibility to certain infectious diseases with skin involvement and some systemic infections. (World Health Organization: WHO/EHG/95.16) In humans, pigmentation appears to provide little protection against UV-induced immune suppression.[5]
Size of population(s) affected	Entire Population
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	L, The science is well developed and defined concerning UV effects on the skin and eyes. UV radiation has been shown to have a suppressive effect on the immune system, but more research into mechanisms is required. There is a need for more research to collect data on the extent and severity of UV exposure in New Jersey in a comprehensive way. Establishment of reporting requirements for UV-induced skin and eye injuries wherever such requirements do not presently exist would help to provide a more inclusive dataset.

<table>
<tr><td>Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description</td><td>H, Additional research is being conducted into the effects of UV on the body and on UV penetration (ozone depletion). There is no question that risk is increasing.[9] The rate of increased risk due to increased solar radiation penetration should be clarified as additional data is obtained.</td></tr>
<tr><td>Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, -, =, where + is improvement)</td><td>(-)Thinning of atmospheric ozone contributes to an increase in solar UV penetration to the Earth's surface. It is expected that ozone depletion, unchecked, will result in an increase in UV exposure and the likelihood of an increase in the negative effects associated with such an increase.
The United Nations Environment Programme has estimated that over 2 million non-melanoma skin cancers and 200,000 malignant melanomas occur globally each year. In the event of a 10% decrease in stratospheric ozone, with current trends and behavior, an additional 300,000 non-melanoma and 4,500 melanoma skin cancers could be expected worldwide. (WHO/EHG/95.16)
As the following table indicates, rates of skin melanomas (Excluding Basal and Squamous) in NJ have generally increased between 1993 and 1996.
<table>
<tr><th rowspan="2">Skin Melanomas (Excluding Basal and Squamous)</th><th rowspan="2">Total Cases</th><th colspan="5">Rates</th></tr>
<tr><th>1993</th><th>1994</th><th>1995</th><th>1996</th><th>1997 Prelim.</th></tr>
<tr><td>Male</td><td>3756</td><td>16.0</td><td>16.2</td><td>16.6</td><td>18.8</td><td>18.7</td></tr>
<tr><td>Female</td><td>2596</td><td>8.3</td><td>10.0</td><td>9.2</td><td>9.7</td><td>11.1</td></tr>
</table>
(Final information for 1997 - 1999 was not provided by the DOH)</td></tr>
<tr><td>Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood</td><td>(L) A catastrophic reduction in atmospheric ozone would allow penetration of significant amounts of UVB and UVC. Due to the interdependent nature of life on the earth, the impact on all plant and animal communities on Earth from the destructive impact of unfiltered UVB and UVC could result in a cascading failure of all ecosystems. In the event that such an event occurred, humanity would find continued existence on Earth to be tenuous It is extremely unlikely that such an event might occur, however the continued depletion of the ozone layer is cause for concern. Given the potentially fatal consequences of a catastrophic ozone depletion, the reduction and/or elimination of ozone depleting chemicals in our environment may be a wise choice.</td></tr>
<tr><td>Extent to which risks are currently reduced through in-place regulations and controls</td><td>Solar Radiation
International and national efforts to reduce atmospheric ozone depletion have met with mixed success. Reductions in ozone depleting emissions will be necessary in order to prevent additional reductions in ozone levels.

Artificial UV Sources
Both the American Medical Association[7] and the American Academy of Dermatology[8] have recommended to the Food and Drug Administration (FDA) that tanning beds be banned for non-medical uses. The FDA has taken the regulatory position of attempting to insure that consumers are adequately notified of the hazards and risks associated with tanning booths.
At the present time, NJ has a statue (NJSA 26:2D-81 "V. Tanning Facilities") which, in part, directs the NJ Department of Health (DOH) to "Establish minimum safety standards for tanning facilities." While this legislation went into effect on April 2, 1990, no action by the DOH to promulgate regulations has been taken.</td></tr>
<tr><td>Relative Contributions of Sources to Risk</td><td></td></tr>
</table>

(H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	L
Small business industry	H (Tanning Salons)
Transportation	N/A
Residential	N/A
Agriculture	N/A
Recreation	N/A
Resource extraction	N/A
Government	L
Natural sources	H
Contaminated sites	N/A
Diffuse and non-NJ sources	
Sediment	N/A
Soil	N/A
Non-local air sources (including deposition)	N/A
Biota sinks	N/A

Human Health Issue Summary: Ultraviolet radiation

What is it?
Ultraviolet radiation is the electromagnetic energy that largely comes from the sun. The stratospheric ozone layer absorbs harmful forms of UV light and depletion of the ozone

Issue: Ultraviolet (UV) Radiation
Author: Robert Kozachek
Version:11/27/00

layer results in increased UV radiation reaching the earth's surface. Ultraviolet radiation is divided into categories based on wavelength; the impacts noted here are associated with ultraviolet radiation known as UV-B. UV-B damages biological systems by causing chemical changes at the molecular level. In humans, UV-B exposure is known to be associated with various skin cancers, accelerated skin aging, cataracts, and other eye diseases. Exposure may also affect the immune system.

What's at risk?

Virtually the entire population of New Jersey is exposed to some level of naturally occurring UV-B daily. People with fair skin are more susceptible to burns and skin cancers than darker skinned individuals. However, eye damage can occur in all populations. Beachgoers and other outdoor enthusiasts are at increased risk, as are workers who spend most of the day outdoors.

What are the human health impacts in New Jersey?

About 6,300 New Jerseyans suffer from skin melanomas, and the Department of Health has documented increases in the incidence of melanoma for the period of 1993 through 1996, the last year final data were available. Individual behavior choices and the reduction in stratospheric ozone may be contributing to these increases. The extent of health effects other than skin cancers is not known. More research is needed to document the extent and severity of UV exposure in New Jersey.

What's being done?

The international "Montreal Protocol" agreement was intended to reduce and eventually eliminate the emissions of man-made substances that deplete stratospheric ozone. The federal Clean Air Act was subsequently amended to include provisions for the protection of the ozone layer. These regulations include a schedule for reducing the production and use of ozone depleting chemicals that is currently being implemented. Education efforts focused on reducing human exposures to ultraviolet radiation help to reduce human health risk.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
Effects on the Skin H3	M3	Y 5	M3
Effects on the Eye H3	H4	N1	M3
Effects on the Immune System L2	L2	N1	L2
			M3

1. The EPA has established a definition of UVB as wavelengths from 280-320nm. The World Health Organization (WHO) measures UVB as between 280 - 315nm.

2. World Health Organization: WHO/EHG/95.17.

3. J. Westerdahl, C. Ingvar, A. Måsbäck, N. Jonsson, H. Olsson, "Risk of cutaneous malignant melanoma in relation to use of sunbeds: further evidence for UV-A carcinogenicity", British Journal of Cancer; p 1593-1599, Volume 82, Number 9, May 2000.

4. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program "9th Report on Carcinogens 2000".

5. A. Screibner, D.E. Hollis, E. Murray, W.H. McCarthy, G.W. Miltion, "Effects of exposure to ultraviolet light on epidermal Langerhans cells and melanocytes in Australians of Aboriginal, Asian and Celtic decent", Photodermatology 3 (1987) 15-25.

6. Data from the New Jersey State Cancer Registry available from 1993 - 1997 only. Information on Basal and Squamous carcinomas is not collected by the New Jersey Department of Health and so is unavailable.

7. American Medical Association policy H-440.937 "FDA Investigating the Safety of Tanning Parlor Devices" (Sub. Res. 415, A-92; Sub. Res. 217, I-94).

8. Letter from the American Academy of Dermatology to the FDA dated July 7, 1999 concerning Docket No. 98N-1170.

9. Journal of Photochemistry and Photobiology B:Biology 1-4 46 (1998) from the 1998 UNEP report.

Issue: VOCs-carcinogenic
Author: Ken Jones, Sue Thomas
Version: 04/30/01

New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	**Volatile Organic Compounds (VOCs) – Carcinogenic**
stressor	

description of stressor (including etiology)	This analysis includes the following chemicals Acetaldehyde Acrylonitrile Hydrazine Ethyl Acrylate Ethylene oxide Each of these chemicals is included in the Cumulative Exposure Project conducted in New Jersey by US EPA. (US EPA (CEP)) The analysis does not include many other chemicals that may be found in New Jersey's environment and could cause cancer, but have not been analyzed. The analysis also does not include acrolein, benzene, 1,3- butadiene and formaldehyde, all carcinogens found at significant levels in New Jersey, but covered in separate reports.
stressor-specific impacts considered including key impacts	Each of these chemicals is included in this analysis because of the possibility of their carcinogenicity. In addition, each of the chemicals has other health effects. Acetaldehyde is an irritant as are other aldehydes, such as formaldehyde and acrolein (US EPA (IRIS)). However, acetaldehyde effects are only observed at levels significantly higher than with formaldehyde. Acetaldehyde is present in the environment based on its use in the chemical industry and as a by-product of combustion, especially from cigarettes (EPA, 1994). In addition to environmental exposure, acetaldehyde is a metabolic byproduct in the breakdown of ethanol. After events of alcohol consumption, circulating acetaldehyde levels in the body are significant, leading to some symptoms of a hangover. Exposure to elevated levels of acetaldehyde in occupational settings can mimic some of the hangover effects such as headache and nausea. (Doull et al., 1980) Acrylonitrile is an irritant affecting the eyes, nose and throat. Regulatory limits based on non-cancer effects are based on inflammation of the epithelium. (US EPA (IRIS))

	Ethyl Acrylate is an irritant affecting the nose and throat. (Doull et al., 1980) Ethylene Oxide can cause lung damage and nervous system damage. Ethylene oxide is present in the environment due to its use in chemical processes and as a fumigant and preserving agent in the food industry. (New Jersey Dept. of Health and Senior Services) Hydrazine is very reactive leading to its role in liver damage and reproductive problems. (Doull et al., 1980)
Exposure Assessment	
exposure routes and pathways considered (include indoor air as appropriate)	This report focuses on the impacts associated with inhalation. Exposures due to ingestion are unknown because of a lack of monitoring for the presence of these chemicals in drinking water.
population(s)/ecosystem(s) exposed statewide	Most of the chemicals in this class vary in concentration significantly due to local variations in emissions. Hydrazine is an extreme case with some areas seeing concentrations 1000 times the amounts noted in other areas based on local production. (NATA)
quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate	This first table provides average statewide air concentrations (in ug/m3), maximum values modeled for counties and compares them to the health benchmarks used in the Cumulative Risk Project (1 x 10^{-6} lifetime cancer risk). (US EPA (CEP)) NJ Aver NJ max 1 x 10^{-6} risk Acetaldehyde 1.21 2.83 (Hudson) 0.45 Acrylonitrile .0007 .0014 (Hudson) .015

	Hydrazine .00015 .0008 (Essex) .0002 Ethyl Acrylate .033 2
	Ethylene Oxide .012 .021 (Middlesex) .01
specific population(s) at increased risk	Risk estimates include the variation in population susceptibility. There is not a subset of the population that is known to be more susceptible to these risks. The authors considered the possibility of characterizing effects on those certain populations more susceptible to the irritant nature of pollutants. The characterization of sensitive populations is described under the topic of multiple chemical sensitivities. This controversial health issue is based upon observations of specific populations that suffer physiological responses from concentrations of pollutants that show no impacts on the general population in clinical studies. One characteristic of exposures that is common in those suffering from Multiple Chemical Sensitivities is the presence of chemicals that surpass an odor threshold. (Kreutzer, Neutra and Lashuay, 1999) Odor thresholds are notorious in their difficulty to determine but of the VOCs considered in this report, only acetaldehyde approaches threshold levels for the most sensitive populations. (Low range threshold = 10 ppb, NATA maximal modeled concentrations= 1.3 ppb) (American Industrial Hygiene Association). For this reason, we do not consider Multiple Chemical Sensitivities for this analysis.
quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations)	
Dose/Impact-Response Assessment	

quantitative dose/impact-assessment employed for each population considered	The health benchmarks for these compounds are all based on their cancer risks. The benchmark is the concentration at which there is an increased risk of one additional lifetime cancer per million population. The non-cancer effects are not apparent at ambient concentrations. A review of data from EPA's Integrated Risk Information System (EPA – IRIS) identifies RfC's for acetaldehyde, and acrylonitrile above the ambient concentrations modeled for New Jersey. Ethylene oxide regulatory limits are established for workplace exposures and are more than 1,000 times the ambient concentrations modeled for New Jersey.
Risk Characterization risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	The total exposures from these chemicals may cause an increase in the number of statewide cancers of 27 over a lifetime or slightly less than one cancer per three years. Most of these cancers are due to effects from acetaldehyde (19 additional cancers over a lifetime) and ethylene oxide (8 additional cancers over a lifetime). These risks are calculated by multiplying the average ambient concentrations of the chemicals modeled under NATA with the risk slope factor and the total population of New Jersey (US Census Bureau). The other chemicals are largely below the health benchmarks and are therefore expected to cause less than one additional cancer over a lifetime.
risk estimate(s) by population at risk, cont.	The rate of cancer increases is higher in Middlesex and Essex Counties by a factor of two from statewide averages based on higher concentrations of acetaldehyde modeled for these counties. A factor of two is much less than other factors effecting exposure to carcinogens such as the mobility of the population and the time averaging of ambient concentrations. The individual increased lifetime rates of cancer generation due to acetaldehyde average about 2.3 per million and range from less than one to about 6.5 per million. For ethylene oxide, there is a state average of slightly more than one additional cancer per million and a maximal value of slightly more than two cases per million. When the effects from the two chemicals are added, the cancer rate averages almost 3.5 per million for the state with the highest rates of about 7.5 per million.
assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Cancer causes death in from one third to 95% of its cases. When death does not result, there is significant pain and trauma due to the cancer and its therapies. (Doll and Peto, 1981)
Size of population(s) affected	The cancer effects from this issue are expected to affect the general population.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	M - As with most cancer causing chemical contamination, there is a great deal of uncertainty regarding the specific impacts from individual chemicals.
assessment of uncertainties in this	M – The studies on these chemicals are not as comprehensive as studies on other chemicals such as dioxin or the halogenated VOCs. The exposure estimates are based on a model that incorporates point source releases and calculated area sources. For other chemicals

assessment (H,M,L) and brief description, and data gaps	undergoing this modeling, actual results from on-the-ground monitors almost always fall within a factor of ten from the modeled results with most results within a factor of three. (NJ DEP, Camden monitoring)
potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	M – There is no expectation that increased research will focus on this group of chemicals. However, the lack of in depth studying suggests that there may be greater room for discovery than with other chemical contaminants. Further research on individual exposures will probably identify a clear range for exposures depending upon occupation and indoor sources.
potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , =̃, where + is improvement)	+ As with most chemicals, general efforts in pollution prevention are resulting in a general decrease in the use and release of VOCs. Comparing results between 1990 and 1996, there is about a 10% reduction in acetaldehyde but a similar increase in ethylene oxide concentrations in New Jersey (NJDEP, CEP). Because there can be wide variations in concentrations on a day to day basis, these small changes should not be weighted too heavily as the basis for determining trends.
potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L – there is a low probability of the release of catastrophic quantities of these classes of chemicals.
extent to which risks are currently reduced through in-place regulations and controls	As with most chemicals, there are general permitting processes in place to reduce the release of VOCs.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
large business/industry	M/H The primary chemicals causing risk are acetaldehyde and ethylene oxide. In the case of acetaldehyde, it has industrial use in the synthesis of acetic acid, but its release is minimal (NATA). Ethylene oxide also has significant industrial use and large sources may be the predominant cause of ambient concentrations.
small business industry	L
transportation	L/M – Acetaldehyde is a significant mobile source product.
residential	L/M – Acetaldehyde is a product emitted from ripe fruits, and in some cases may be a significant source. (US EPA, 1994)
agriculture	L (Although ethylene oxide has a significant use in food processing)
recreation	L

resource extraction	L
government	L
natural sources	L
contaminated sites	L
diffuse and non-NJ sources	
sediment	L
soil	L
non-local air sources (including deposition)	L
biota sinks	L/M Acetaldehyde has significant natural sources

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	**Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)**	**Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)**	**Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)**
Cancer 5	3	3	2
			2

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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	**Volatile Organic Compounds (VOCs) – Non- carcinogenic**
Stressor	
Description of stressor (including etiology)	This problem area includes the following chemicals: Toluene Xylene Methanol Methyl ethyl ketone Glycol ethers Toluene and Xylene are largely present as the result of use of petroleum fuels. Methanol is a common chemical feedstock, and solvent with many commercial uses. Methyl ethyl ketone is used in many industrial processes. The most common glycol ether is ethylene glycol which is a common antifreeze and solvent.
stressor-specific impacts considered including key impacts	(We focus on low concentration impacts as high concentrations are not important in considering environmental exposures.) Toluene and xylene at low concentrations can effect liver function and have neurologic effects including narcotic effects. (US EPA (IRIS)) Ethylene Glycol can irritate the eyes nose and throat. Ingestion can lead to headache, nausea and vomiting. (Doull et al., 1980) Methyl Ethyl Ketone has neurologic effects including memory effects, mood changes, malaise and altered sleep patterns. (New Jersey Department of Health and Senior Services) Methanol is an irritant to the eyes, mouth and throat and can damage the liver. (New Jersey Department of Health and Senior Services)

Exposure Assessment	The primary focus of this report is on inhalation due to air emissions. There are also impacts from ingestion via drinking water, but these compounds are rarely reported to be present in drinking water at levels that cause known effects. While Xylene and Toluene are often detected in drinking water, there have not been any exceedance of regulatory limits (MCL) since 1993. (personal communication, Sandy Kreitzman, New Jersey DEP) Accidental exposures may be much more important, such as poisoning via drinking antifreeze, but this report does not investigate these accidental exposures.
Exposure routes and pathways considered (include indoor air as appropriate)	The primary route of exposure considered in this analysis is via inhalation. There are two sets of conditions important in measuring inhalation exposure. Outdoor exposure represented by ambient air quality monitoring and indoor exposures represented by individual monitoring.
Population(s)/ecosystem(s) exposed statewide	The general population is exposed to low concentrations of these chemicals while out of doors. In some cases, indoor concentrations are higher. Workers are exposed to higher concentrations and are, in fact, the basis for most regulatory activity regarding their airborne concentrations.
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	The table below includes the highest modeled concentrations for some of these chemicals reviewed in the Cumulative Exposure Project. (US EPA, (CEP)). These represent ambient outdoor conditions. Toluene 3.8 ug/m3 Xylene 2.9 ug/m3 Methanol 1 ug/m3 All of these figures are estimates based on a model, which includes point source, area source and mobile sources of emissions. The estimates can be compared with on-the-ground monitoring sites such as Camden site, in place for several years. When comparing the results from on-site monitoring to results from models, there is general agreement (within a factor of two or three) although there are wide temporal fluctuations evident at the monitoring sites that are not apparent in the averages reported from the model. For indoor exposures, we rely on results taken from a study in the 1980s. In that study, xylene and toluene are noted for their elevated concentrations. (Indoor Air Quality Information Clearinghouse) M and p-xylene 21-29 ug/m3 Toluene 60-62 ug/m3
	We rely on a report from Richard Kreutzer to identify a proportion of the population that may be susceptible to effects to trace chemical contaminants through multiple chemical sensitivity. (Kreutzer et al.) While this effect is controversial, there is evidence that some biological functions are altered under exposure to trace contaminants. One key to symptoms of multiple chemical sensitivities is the relation of ambient conditions to an odor threshold. In the case of xylene and toluene, the minimum odor thresholds reported are 170 ppb or at least ten times the reported concentrations in the 1980s indoor air quality studies (American Industrial Hygiene Association).

Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations)	The exposure for indoor air pollutants is difficult to characterize. Newer buildings, generally have greater exposures to chemicals; but for xylene and toluene, the presence of an attached garage or the recent use of paints and caulks promotes greater indoor concentrations (Indoor Air Quality Information Clearinghouse).
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	Most studies on these compounds determine the potential for health effects at concentrations greater than 100 parts per million. From these studies, occupational standards are established. In no case, do the concentrations in ambient air approach 1 part per million. For indoor air, average concentrations are still well below a 1 ppm level. However, there can be cases with indoor concentrations approaching 1ppm (Indoor Air Quality Information Clearinghouse). In summary, the risks do not approach those of other indoor air pollutants such as formaldehyde, ozone, nitrogen dioxide and molds. The remaining potential for effects is among those that are extremely sensitive to low levels of pollutants. However, the odor threshold for these chemicals is typically above the concentrations found in indoor air (American Industrial Hygiene Association).
Risk Characterization Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed, e.g., mean population risk upper percentile population risk, etc.)	For this issue, we can presume negligible risk for general populations. The only possibility for harm from ambient concentrations of these chemicals is for populations susceptible to Multiple Chemical Sensitivities. Even for this population, these non-halogenated compounds are unlikely to trigger reactions.
Risk estimate(s) by population at risk, cont.	
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	
Size of population(s) affected	
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	L/M - It is possible that new information will uncover damages previously unexpected, but occupational studies have been in place for several years and the occurrence of new effects is decreasing. There are several chemicals that have not been the subject of intensive study. However, there is little epidemiological evidence to suggest that new research will uncover new health effects.
	L

Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , ~, where + is improvement)	+ There are general decreases in the uses of organic solvents and petroleum refueling facilities increase the recapture of vapors.
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L - These chemicals do not cause acute health effects at low concentrations. Their volatile nature results in rapid dispersion after release suggesting that individuals will probably not be exposed to dangerous concentrations.
Extent to which risks are currently reduced through in-place regulations and controls	These chemicals are not subject to in-depth regulation, rather they are regulated in parallel with other manufacturing and petroleum transfer activities.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	.
Large business/industry	L/M The chemical industry still releases significant amounts of these chemicals. For xylene and toluene, Major Source Emissions represent a little more than a 5% of the total (US EPA (NTI)).
Small business industry	L
Transportation	M/H Xylene and Toluene are found in motor vehicle fuels and released during refueling and inefficient combustion. More than two thirds of the state totals result from transportation sources (US EPA (NTI)).
Residential	L
Agriculture	L
Recreation	L
Resource extraction	L
Government	L
	L

Natural sources	
Contaminated sites	L
Diffuse and non-NJ sources	
Sediment	
Soil	
Non-local air sources (including deposition)	L
Biota sinks	

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
Out door exposures 1-L			1-L
Indoor exposures 2-L/M	2-L/M	2-L/M	2-L/M
			2-L/M

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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	
Stressor	**Waterborne Pathogens** This review covers all waterborne pathogens except *Cryptosporidium* and *Legionella* which are covered under separate risk assessments (Atherholt, 2000a; Atherholt, 2000b).
Description of stressor (including etiology)	Most but not all waterborne disease-causing microorganisms are enteric pathogens. Enteric pathogens are present in the feces of infected humans or animals. Waters used for drinking or recreational activities occasionally become contaminated with feces containing enteric pathogens. There are three main groups of these pathogens; bacteria, viruses and parasites. The parasite group includes the single celled protozoans and multicellular organisms including some flatworms and roundworms. Enteric bacteria contributing to US disease outbreaks between 1986-1998 include *E. coli* (mostly strain O157:H7), *Shigella* (usually *S. sonnei*), *Salmonella*, *Leptospira*, and *Campylobacter* (Barwick *et al.*, 2000; Herwaldt *et al.*, 1991; Kramer *et al.*, 1996; Levine and Craun, 1990; Levy *et al.*, 1998; Moore *et al.*, 1993). Other enteric bacterial pathogens include *Klebsiella, Serratia,* some *Vibrio* spp. and *Yersinia* (AWWA, 1999; Hurst *et al.*, 1997). Enteric viruses contributing to US disease outbreaks include caliciviruses (including Norwalk and Norwalk-like viruses), adenoviruses, and hepatitis A virus. Other enteric viral pathogens include astroviruses, enteroviruses (echoviruses, Coxsackie viruses), hepatitis E virus, reoviruses, and rotaviruses. Enteric parasites contributing to US disease outbreaks include; *Cryptosporidium parvum* (Crypto), *Giardia lamblia*, and members of the Schistosomatidae. Other enteric parasite pathogens include *Toxoplasma gondii, Ascaris lumbricoides, Balantidium coli, Blastocystis hominis, Cyclospora cayetanensis, Entamoeba, Isospora belli*, microsporidia, *Taenia solium*, and *Trichuris trichiura*.

Description of stressor (including etiology) (continued)	Some waterborne pathogens are not enteric pathogens. Such pathogens can cause skin, wound, eye, ear, nose, throat or lung infections. Some of these pathogens may be derived from contaminated feces but others come from either the skin or exudates of nearby infected persons. Still others are normal inhabitants of soil or water. The most common non-enteric pathogen in the U.S. disease outbreak data (36 outbreaks of dermatitis between 1991-1998) is the bacterium *Pseudomonas aeruginosa* which is a problem organism in spas and hot tubs (Barwick *et al*, 2000; Kramer *et al.*, 1996; Levy *et al.*, 1998; Moore *et al.*, 1993). Other non-enteric bacterial pathogens include *Legionella, Acinetobacter, Aeromonas, Flavobacterium, Klebsiella, Mycobacteria, Pleisiomonas shigelloides, Staphylococcus* and some *Vibrio* spp. Non-enteric protozoans and multicellular parasites include *Naegleria* (which causes a rare, but fatal meningoencephalitis) and *Acanthamoeba* (AWWA, 1999; Hurst *et al.*, 1997). Note: Pfiesteria are a group of estuary pathogens of fish. Pfiesteria's adverse affect on fish is through the release of one or more potent toxins. Humans exposed to these toxins, via inhalation of aerosols or skin contact with Pfiesteria toxin-containing water, have been shown to have a variety of adverse health effects. However, this organism is not able to infect (multiply within) humans. Separate human health and ecological quality technical workgroup documents for Pfiesteria are available.
Stressor-specific impacts considered including key impacts	Some pathogens have a low level of virulence. They either are not able to infect a majority of exposed persons or else they can cause an infection (multiplication within the host) without causing illness (adverse clinical symptoms) in most of the infected persons. Other pathogens are more virulent. They can cause illness, sometimes severe, in a majority of infected individuals. Many pathogens have low levels of virulence in the general population but higher levels of virulence in various sensitive sub-populations (see below). Many illnesses are mild (*e.g.,* gastroenteritis, which can be caused by a variety of pathogens) but some are quite serious. The more serious diseases (and the disease-causing agents) include meningitis, encephalitis, myocarditis, paralysis (echoviruses and Coxsackie viruses), hepatitis and liver failure (hepatic A virus), hemolytic uremic syndrome (some strains of *E. coli* and *Shigella*), ulcers and stomach cancer (*Helicobacter pylori*) and serious wound infections (*Aeromonas* and *Vibrio*). Some disease agents can cause mortality (*e.g.*, *Legionella, E. coli, Naegleria*). Some pathogens cause serious disease only in sensitive sub-populations such as persons with underlying disease or immunosuppression, infants, the elderly, surgical patients or those with invasive equipment or those undergoing antibiotic therapy. Examples of such microbes include *Acinetobacter, Aeromonas, Mycobacteria*, some *Pseudomonas, Serratia*, adenoviruses and rotaviruses (affecting primarily young children), *Cryptosporidium* and microsporidia (see below).

	Many serious diseases of the past, caused by virulent pathogens, are only found today in developing countries. Their absence in North America and European countries is due to the advent of potable water filtration and disinfection, improved sanitation practices, vaccines, and antibiotics. Such historic diseases (and the causative agent) include poliomyelitis (poliovirus), amoebic dysentery (*Entamoeba histolytica*), cholera (*Vibrio cholerae*), and typhoid fever (*Salmonella typhi*).
Exposure Assessment	
Exposure routes and pathways considered (include indoor air as appropriate)	Ingestion or contact with contaminated water. The most common disease route is the fecal-oral route in which pathogenic organisms multiply in the intestines of infected (but not necessarily ill) people or animals. They are then shed in the feces which can enter surface or ground waters from septic tanks, sewage treatment plants or broken or leaking sewage collection pipes. Feces from infected pets, farm animals and wild animals can pollute waters directly or from storm water runoff. These waters are then consumed, without treatment or with inadequate treatment, by other susceptible (not immune) individuals. Contaminated water can also make a person sick by first contaminating hands (or objects followed by hand contact). Contaminated hands then come into oral contact to complete the fecal-oral route of transmission. Ingestion can be deliberate (drinking water) or inadvertent (during swimming or other watersport activities). Foodborne infection can occur from the waterborne fecal-oral route through the consumption of contaminated shellfish (which concentrate pathogens from surrounding waters) or from consumption of crops which have been contaminated by domestic animal wastes or following irrigation using contaminated water. Infection can also occur following exposure to non-fecal pathogens. Some pathogens are natural inhabitants of soil and water and hence are not related to the presence of fecal pollution. Other non-fecal pathogens can be transmitted by person-to-water-to-person contact following shedding from the skin or exudates of infected persons.
Exposure routes and pathways considered (continued)	Drinking Water Federal and state laws (see below) require filtration and disinfection of potable water. Filtration and disinfection eliminate all but very low levels of a few types of potentially pathogenic microbes. Pathogens most likely to survive the conventional water treatment process, and hence of most public health concern, are small (most able to pass through filters), disinfectant-resistant organisms. Disinfection-resistant bacteria include *Mycobacteria* and *Legionella* (Sobsey, 1989). Disinfectant-resistant viruses include caliciviruses, rotaviruses, and hepatitis A virus (Keswick *et al.*, 1985; Sobsey, 1989). Unfortunately, many protozoan pathogens, *Cryptosporidium* in particular, are disinfectant-resistant (AWWA, 1999).

	Because they are the smallest of the pathogen groups, viruses have the highest likelihood of passing through granular filters that are used in most water treatment plants. Properly treated drinking water is usually free of disease-causing organisms, including disinfectant-resistant ones, or contains them at levels thought low enough to be safe, but this is not known for certain. Treated water may be an occasional infection source due to low levels of these organisms or due to treatment deficiencies or post-treatment contamination due to pipe ruptures.
Exposure routes and pathways considered (continued)	Recreational waters: Several state and federal regulations also help protect state recreational waters (see below). Despite these regulations, low levels of pathogens may be present in untreated waters. Most pathogens, if present, are derived from fecal contamination but others are normal inhabitants of surface waters (*e.g.*, *Vibrio* spp. in marine waters, *Legionella* in fresh waters). Exposure to enteric pathogens in untreated surface waters such as lakes, rivers and reservoirs can occur during swimming or participation in watersport activities, following inadvertent ingestion of contaminated water. Infection can also occur by purposeful ingestion of untreated surface water or contaminated ground water. Exposure to non-enteric pathogens can occur following immersion in a waterbody. Thus, potential exposure to low levels of pathogens in untreated waters, even those meeting NJ's surface water quality criteria for fecal indicator bacteria, is unavoidable. State regulations are also in place to prevent pathogen exposure in treated recreational waters (see below). Despite this, chlorinated swimming, wading and wave pools are occasional sources of infection by disinfection-resistant organisms (*e.g.*, *Cryptosporidium*), particularly when used by diaper-age children (Barwick *et al.*, 2000; Levy *et al.*, 1998). For both treated as well as untreated recreational waters, disease transmission can occur by the fecal-oral route (see above) and by immediate person-to-water-to-person transmission in high bather-density waters (Barwick *et al.*, 2000; Levy *et al.*, 1998).

Population(s)/ecosystem(s) potentially exposed statewide Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	Everyone in the state (8.4 million) is potentially exposed through either drinking contaminated water or by swimming or conducting watersport activities. Infection during recreation can occur in marine or fresh waters, including swimming pools and water parks. Actual number of persons so exposed is unknown and can vary greatly from year to year. Drinking water: Concentrations of waterborne pathogens in treated drinking water are not known. Nevertheless, concentrations are expected to be very low. For example, for *Cryptosporidium*, an average concentration in treated water is estimated to be not more than 1 oocyst per 11,200 liters (Atherholt, 2000a). Concentrations of other pathogens are expected to be even lower or absent altogether.
	Monitoring directly for pathogens is problematic for several reasons. Pathogens are not always present in fecal wastes. Different pathogens may be present at different times. When present, they may occur at low levels, which are often difficult to measure. Many pathogens are difficult or impossible to grow in the laboratory. Finally, many pathogen tests are difficult, expensive and time-consuming. For these reasons, the potential presence of enteric pathogens in water is indicated by the presence of certain fecal-associated bacteria or bacterial groups. Indicator bacteria are always present in fecal wastes, the source of enteric pathogens, and are easy to measure. The indicator bacteria monitored for drinking water are total coliform (TC) bacteria. Treated drinking waters must be essentially free of coliform bacteria at all times (see below). There is concern among scientists and regulators that there may be pathogens (*i.e.*, some viruses and protozoa) that are more resistant to filtration and disinfection than the bacterial indicators. Hence, some pathogens may be present in the absence of indicator bacteria. Therefore, there is current research ongoing in NJ and worldwide to try to identify a reliable indicator for viral and protozoan pathogens.
Quantification of exposure levels statewide, including populations at significantly increased exposure (continued)	Recreational waters: The concentration of pathogens in recreational waters is not known. They are not always present. When present, concentrations can vary greatly in time and space. Because pathogen monitoring is problematic (above), the concentration of enteric pathogens in recreational water is estimated by the concentration of indicator bacteria. The current regulation relies on concentrations of fecal coliform (FC) bacteria (N.J.A.C. 1996; see below), but the indicator will change by the year 2003 to enterococci (Ent) monitoring for marine bathing beach waters and either enterococci or *E. coli* for fresh waters (BEACH 2000; USEPA, 1986; USEPA, 1999). These criteria ensure that pathogens, if present, are present at levels thought low enough to be safe. However, pathogens may be present even in waters meeting the regulatory limit. There is currently no indicator for non-enteric pathogens.

Specific population(s) at increased risk	Depending on the pathogen, populations at increased risk (and example causative disease agents) include: Persons with underlying disease (*Pseudomonas*). Persons with weakened immune systems (*Mycobacteria, Pseudomonas, Cryptosporidium*, microsporidia, *Toxoplasma*). Infants (some *E. coli*, *Pseudomonas*, adenovirus, enteroviruses, rotavirus). Elderly (*Klebsiella, Pseudomonas*). Pregnant women (hepatitis E virus). Surgical patients or those with invasive equipment (*Klebsiella*). Persons undergoing antibiotic therapy (*Aeromonas*). All of the above (*Acinetobacter*). Combined, these sensitive sub-populations represent 20-25 % of the U.S. population (ASM, 1999). For NJ (assuming an equal distribution of sensitive people throughout the US) this would represent 1.6 to 2 million people.
Quantification of exposure levels to population(s) at increased risk (*i.e.*, susceptible sub-populations) (include indoor air as separate category as appropriate)	Unknown but not different than that of the general population.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	Waterborne disease estimates from the Centers for Disease Control and Prevention (Bennett *et al.*, 1987).
Risk Characterization	
Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , *e.g.*, mean population risk upper percentile population risk, etc.)	None used.

Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	The adverse health effects from most pathogens are comparatively mild. Symptoms such as diarrhea, cramps, nausea and sometimes vomiting are common symptoms of many enteric pathogens. However, some illnesses are serious. Encephalitis, myocarditis, hepatitis, and paralysis are all serious consequences of infection by some enteric pathogens. There is some mortality associated with disease outbreaks caused by other waterborne pathogens such as *E. coli* O157:H7, *Shigella*, *Legionella* and others (see above).
Size of population(s) affected	In 1987 it was estimated, based upon data collected by the U.S. Centers for Disease Control and Prevention, that there were approximately 940,000 cases of illness and 900 deaths each year in the U.S. from waterborne diseases (Bennett et al., 1987). U.S. census figures released 12/28/00 show the April 1, 2000 NJ resident population at 8,414,350 and the total April 1, 2000 U.S. resident population at 281,421,906 (**http://www.census.gov)**. Therefore in the U.S., waterborne diseases affect an estimated one out of 300 people with one death for every 313,000 people. New Jersey's annual share of the U.S. waterborne disease burden, based on current census data and disease estimates of Bennet *et al.,* (1987), and assuming a uniform geographic distribution of cases, is 28,000 illnesses and 27 deaths.
	This estimated disease burden, if accurate, goes largely undetected. For every waterborne disease outbreak which is detected, there are other outbreaks which go undetected due to the fact that symptoms for the most part are not serious (*e.g.*, gastroenteritis and symptoms which mimic diseases acquired from other exposure routes) and most ill individuals do not seek medical attention. An outbreak is defined as clinical illness in 2 or more people resulting from a common exposure to a water source contaminated by a disease-causing microbe. In addition, for every disease outbreak (detected or undetected), there is a larger amount of endemic disease. Endemic disease is defined as indigenous disease peculiar to a particular people or location. In this case, it is essentially ongoing but undetected cases of waterborne-disease not related to common exposures. It is primarily endemic disease that affects the above estimated number of NJ citizens each year.
Size of population(s) affected (continued)	NJ has not had a documented drinking water-related microbial disease outbreak since 1989 (although high nitrite levels affected 6 people at an office in 1996 [Levy *et al.*, 1998]). The 1989 outbreak was from a contaminated well at a campsite (Herwaldt *et al.*, 1991). Eight persons were infected with a pathogen which was not identified. A water treatment deficiency was noted as the cause of the outbreak. NJ has had 6 documented waterborne disease outbreaks in recreational waters since 1989 (Levy *et al.*, 1998; Kramer *et al.*, 1996; Herwaldt *et al.*, 1991). All of these outbreaks occurred during the summer, in lakes used for swimming, and affected an average of 140 (range 17 - 418) people. The causative agents for these outbreaks included *Cryptosporidium*, *Giardia*, *Shigella*, *Shistosoma* and 2 outbreaks of unknown etiology.

Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	?? The methods employed to derive the waterborne illness and death estimates are not specified in the article by Bennett *et al.,* (1987). (The article does mention an appendix available from the Carter Center in which data details are provided.) Furthermore, since the waterborne disease estimates are from 1985 and earlier data, it is possible that the current annual disease burden may be different.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	Low. There is no reason to suspect that more accurate waterborne disease data will become available in the near future.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, □, =, where + is improvement)	Waterborne contaminants are largely the result of natural biological processes. In the absence of significant new investment in source water purification, the occurrence of pathogens in drinking water is not likely to change substantially.

Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	Drinking Water Low, but possible. The potential for a waterborne disease outbreak affecting a large number of people in a short period of time (days to weeks) is considered to be low but possible. In 1993, a waterborne disease outbreak in the city of Milwaukee, Wisconsin, caused by the consumption of inadequately treated drinking water contaminated with *Cryptosporidium parvum*, affected an estimated 403,000 people (MacKensie *et al.*, 1994). As many as 4,000 of these people required hospitalization and the disease burden may have contributed to as many as 100 deaths in persons with weakened immune systems (Morris *et al.*, 1996). Four other outbreaks in the U.S. and Puerto Rico between 1991-1998, caused by *Cryptosporidium*, *Giardia*, or of unknown etiology, each affected over 1,000 people. There were 6 other U.S. outbreaks each affecting over 300 people (caused by *Cryptosporidium*, *Giardia*, *Salmonella*, or of unknown etiology [3])(Barwick *et al.*, 2000; Levy *et al.*, 1998; Kramer *et al.*, 1996; Moore *et al.*, 1993). Several large potable water treatment plants operate in NJ with customer populations greater than 100,000. If a treatment breakdown occurred in combination with a pathogen contamination event at any of these plants, a large number of people could be adversely affected. Recreational Water Low, but possible. Two recreational waterborne disease outbreaks in the U.S. each affected over 1,000 persons (Levy *et al.*, 1998). Both of these outbreaks, due to *Cryptosporidium*, occurred in chlorinated pools in water/amusement parks. There have been 7 other U.S. outbreaks each affecting 300 or more people (caused by *Cryptosporidium* [3], *Shigella*, *Leptospira*, adenovirus, or of unknown etiology)(Barwick *et al.*, 2000; Levy *et al.*, 1998; Kramer *et al.*, 1996; Moore *et al.*, 1993).
Extent to which risks are currently reduced through in-place regulations and controls	Drinking water: Most public water purveyors in New Jersey are required to disinfect their water (NJAC 7:10-4.1; USEPA, 1989a) and all public supplies which derive their water from surface sources such as lakes and rivers must also filter their water to reduce viruses, *Giardia* and *Cryptosporidium* in their source water by 99.99%, 99.9% and 99% respectively (NJAC 7:10-9; USEPA, 1989a; USEPA, 1998; USEPA 2000a). The *Cryptosporidium* reduction rule is not yet final for systems serving less than 10,000 people (USEPA, 2000a). Ground waters are protected by soil filtration and by state and existing (SDWA, 1996; USEPA, 1997) and proposed (USEPA, 2000b) federal regulations keeping pollution sources away from public wells. There are no current laws protecting private wells, but a newly passed NJ law will require testing of a private well for pathogen indicator bacteria (see below) as well as other parameters upon the sale of a residence. Landlords with private wells will be required to conduct such testing every 5 years (NJ, 2000). Together, filtration and disinfection eliminate all but very low levels of a few types of potentially pathogenic microbes (see above).

	Monitoring for pathogens is problematic (see above). Thus, the presence of enteric pathogens in treated drinking water is indicated by monitoring for the presence of indicator bacteria. Treated water from all public water treatment systems must be monitored for fecal indicator bacteria (NJAC 7:10 -5 & -7.2; USEPA, 1989b). The indicator bacteria monitored for drinking water are coliform bacteria. The number of required tests depends on the size of the population the water treatment plant serves and on past coliform test results. The required test frequency varies from 1 test every 3 months for the smallest plants to a maximum of 480 tests per month for the largest plants. Treated drinking waters must be essentially free of coliform bacteria at all times.
Extent to which risks are currently reduced through in-place regulations and controls (continued)	Recreational Waters The concentration of enteric pathogens in recreational water is estimated by the concentration of indicator bacteria. The current regulation for designated bathing beaches relies on concentrations of fecal coliform bacteria (N.J.A.C. 8:26-7.19). Based on weekly sampling during the bathing season, all bathing beach water samples must have fecal coliform counts less than 200 colony forming units (cfu) per 100 ml sample. A count of 200 cfu/100 ml or more, followed by another count of 200 cfu/100 ml the next day results in mandatory closing of that designated bathing beach until a < 200 cfu/100 ml count is obtained and a sanitary survey is satisfactory. Most surface waters in the state (lakes and streams) that are listed for primary contact recreation (*e.g.*, wading, swimming, diving, surfing, water-skiing), but which are not bathing beach locations *per se*, have criteria for fecal indicator bacteria (NJAC 7:9B). Two indicator bacteria, fecal coliforms (FC) and enterococci (Ent) are specified. The criteria indicated are geometric mean values of 5 or more samples equally spaced over a 30 day period (samples collected during the summer). For FC bacteria, the criteria for saline coastal waters within 1500 feet of the shoreline are 50 colony forming units (cfu) per 100 ml. The value for all other saline waters and for all such fresh waters is 200 /100 ml and not more than 10% of these samples can exceed 400/100 ml. For enterococci, the criteria for all saline waters are 35 cfu / 100 ml, nor shall any single sample exceed 104 cfu / 100 ml. For fresh waters, the criteria are 33 cfu / 100 ml, nor shall any single sample exceed 61 cfu / 100 ml.
Extent to which risks are currently reduced through in-place regulations and controls (continued)	NJDHSS regulations are also in place to protect swimming pools (including wading and wave pools) and hot tubs and spas from pathogens (NJAC 8:26). Disinfection and disinfectant concentrations are specified for swimming pools (8:26-3.22 & -7.8) and hot tubs and spas (8:26-4.8 & -7.12). Swimming pools and hot tubs and spas may not have heterotrophic plate count bacteria at levels greater than 200 colonies per ml (8:26-7.6 & -7.11). Swimming pools must also not have measurable levels of total coliform bacteria (8:26-7.6) and hot tubs and spas must also not have measurable levels of *Pseudomonas aeruginosa* (8:26-7.11). Like *Legionella*, *Pseudomonas* is a potential pathogen in hot water systems.
Relative Contributions of Sources to Risk (H,M,L)	

Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	Low. Septic tank effluents and treated or improperly treated domestic wastes from industrial sources.
Small business industry	Low. (Same as large business).
Transportation	None.
Residential	Medium. Septic tank effluents and pet animal wastes.
Agriculture	Medium. Some domestic animal wastes may contain high levels of pathogens (*e.g.,* cattle, chickens and turkeys). Also, high levels of nutrients in storm water run-off from farms may contribute to the growth of potentially toxic algae or pathogenic bacteria.
Recreation	High. Many disease outbreaks and presumably a comparatively high level of endemic disease occurs following primary contact recreational activities such as swimming. Numerous outbreaks have occurred in chlorinated swimming pools and water parks in addition to untreated surface waters such as lakes and reservoirs (Barwick *et al.*, 2000; Levy *et al.*, 1998; Kramer *et al.*, 1996; Moore *et al.*, 1993; Herwaldt *et al.*, 1991; Levine and Craun, 1990).
Resource extraction	None.
Government	High. Most sewage treatment plants are run by regional, county or local government entities (although privatization is an increasing trend). Treated or improperly treated domestic wastes may contain waterborne pathogens. In addition, government entities are often the owners of storm drains and combined sewer discharges which are also sources of

	waterborne pathogens.
Natural sources	Low (see biota below). Some pathogens are free-living in waterbodies and are not associated with pollution.
Contaminated sites	None.
Diffuse and non-NJ sources	Medium. Storm water contains high levels of fecal indicator bacteria and thus may contain waterborne pathogens. Most fruits and vegetables consumed during the winter are imported from Mexico and Latin America. Because of inadequate wastewater treatment, irrigation waters used in many of these countries contain pathogens. Several disease outbreaks have originated from such sources (ASM, 1999).
Sediment	Medium. Run-off that occurs following storm events typically contains resuspended sediments from river bottoms and storm drains. Such sediments typically contain high levels of fecal indicator bacteria and hence the potential presence of waterborne pathogens.
Soil	Low. A few types of pathogens (*e.g., Ascaris*) are adapted to survive under dry conditions and may be found in soils, if they are impacted by animal wastes, but most waterborne pathogens do not survive in soils for very long periods of time.
Non-local air sources (including deposition)	None.
Biota sinks	High. Humans, domestic and indigenous animals are the ultimate sources of most, but not all waterborne pathogens.

Human Health Issue Summary: Waterborne Pathogens

What is it?

Bacteria, viruses, and parasites that are present in the feces of infected individuals can contaminate surface waters that may be used as sources of drinking water or for primary

Issue: Waterborne Pathogens
Author: Tom Atherholt
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contact recreation. Waterborne pathogens contributing to disease outbreaks in the U.S. include the bacteria Shigella, Salmonella, Leptospira, and Campylobacter; viruses caliciviruses, adenovireuses, and hepatitis A; and the parasite Giardia. (Cryptosporidium and legionella are addressed separately.) The health effects from waterborne pathogens are generally mild, and may include diarrhea, cramps, nausea, and vomiting. Infections can sometimes result in more serious illness, or even death, particularly among sensitive populations.

What's at risk?

Everyone in New Jersey is potentially exposed via either contaminated drinking water or accidental ingestion while participating in water sports. While no more likely to become exposed, some individuals may be at increased risk for more serious health effects. These include people with weakened immune systems or underlying disease, pregnant women, infants, and the elderly. This sensitive population is estimated at 1.6 to 2 million individuals.

What are the human health impacts in New Jersey?

U.S. Centers for Disease Control data suggest that waterborne pathogens in New Jersey may result in approximately 28,000 illnesses and 27 deaths annually. However, many of these would go largely undetected, because symptoms are typically not serious or distinguishable from other potential sources of illness. New Jersey has not had a documented drinking water related disease outbreak since 1989 when 8 individuals were infected as a result of a contaminated well at a campsite. There have been 6 incidences of waterborne disease as a result of recreational exposures. There is a low risk of a large-scale disease outbreak in the event of a treatment breakdown at any of New Jersey's large drinking water facilities. If this should occur during a pathogen contamination event, a large number of people could be infected.

What's being done?

Disinfection and filtration of water supplies derived from surface water sources eliminate all but very low levels of most pathogens. Testing requirements vary from once every 3 months up to 480 tests per month depending on the size of the facility. New legislation will require testing of private wells for indicator bacteria upon the sale of a residence, and landlords will be required to test every five years. Recreational waters are sampled for indicator bacteria—on a weekly basis for designated swimming areas, or as part of the state surface water monitoring program for lakes and streams that are designated as primary contact recreational waters.

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
Drinking Water			
L, 1	L - M, 2	Y, 5	L, 1

Recreational Water			
L-M, 2	M-H, 4	Y, 5	M, 3
			DW: L, 1 RW: M, 3

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Issue: West Nile Virus
Author: Terry Schulze
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New Jersey Comparative Risk Project
Human Health TWG
Stressor-Specific Risk Assessment

Risk Assessment Framework	Findings
Hazard Identification	**West Nile Virus**
Stressor	West Nile Virus (WNV), Family Flaviviridae, is a mosquito borne virus in the Japanese encephalitis antigenic complex. Two other mosquito borne viruses of importance in NJ, Eastern equine encephalitis virus (EEE) and St. Louis encephalitis virus (SLE), have not caused human disease in the state in over 10 years and are not reviewed here.
Description of stressor (including etiology)	WNV is distributed in a wide geographic range including Africa, the Middle East, West Asia and Europe. In 1999, WNV was identified in North America for the first time, during an epidemic in the New York metropolitan area. WNV has emerged as a significant threat to human, equine and wild bird health in New Jersey and the entire northeastern U.S. WNV is an arthropod-borne virus (arbovirus) that is maintained in a complex zoonotic cycle of biological transmission between infected wild bird hosts. In NJ, the principal enzootic vector mosquitoes are *Culex pipiens* and *Cx. restuans.* Humans and equines may become infected by the bite of secondary, or bridge vector mosquitoes. Humans and equines are not involved in the transmission cycle.
Stressor-specific impacts considered including key impacts	Most infections produce no symptoms in people, or are mild or moderate. Symptoms may include fever, headache, and body aches, often with skin rash and swollen lymph glands. More severe infections may be marked by headache, high fever, neck stiffness, muscle weakness, stupor, disorientation, convulsions, paralysis, coma, and, rarely, death. In the Northeast US there were a total of 21 cases (2 deaths) in 2000. In NJ in 2000, there were 6 confirmed cases of severe WNV disease, including 1 death, from 5 counties. Since most infected people are asymptomatic, the fatality rate is less than 1%. The case fatality rate for hospitalized patients may range from 3% to 15%, and are highest in the elderly. 28 equine cases of WNV were confirmed in NJ in 2000 from 11 counties. 8 horses either died or had to be euthanized. There was a major economic impact on the equine industry from cancelled international events, etc. Avians serve as the reservoir hosts for WNV and are variably impacted by infection. Crows and other corvids are particularly susceptible to WNV infection, and it is estimated that over 95% of the infected crows die. 1,283 birds (mostly crows) were confirmed positive for WNV in NJ in 2000. Crows have proven to be a valuable sentinel animal to monitor WNV activity. There is concern with mortality in hawks, falcons, and zoo birds. Over 70 species of birds in the US have been found to be infected, but the impact on bird populations is not known.
Exposure Assessment	

Exposure routes and pathways considered (include indoor air as appropriate)	All human cases of WNV in the US have resulted from the transmission of WNV from the bites of vector mosquitoes that have become infectious after feeding on viremic birds. The incubation period in the mosquito is from 10 days to 2 weeks after feeding on the infected bird, after which it can transmit the virus to another bird, human or other animal when it takes another blood meal. There is no risk of human-to-human transmission of WNV. In Africa and Russia, ticks may be secondary vectors, but to date only mosquito vectors have been identified in the US. Environmental factors favoring *Culex spp*. mosquitoes could enhance the bird-mosquito enzootic cycle. Rainfall events in August resulting in large broods of floodwater *Aedes spp*. could result in increased human risk.
Population(s)/ecosystem(s) exposed statewide	Everyone in the state is potentially exposed to the bite of an infectious mosquito. The risk is extremely low (6 cases of WNV per 8.4 million population in 2000). Personal protection from mosquito bites and mosquito source reduction and control can further reduce risk. Equines are at relatively higher risk than humans (28 cases in 2000), but an equine vaccine for WNV may be available in 2001. There is a major concern with susceptible wild bird populations potentially impacted by WNV (see above), particularly crows, blue jays, hawks and falcons.
Quantification of exposure levels statewide, including populations at significantly increased exposure (include indoor air as separate category as appropriate)	In 1999 and 2000, a majority of human cases of WNV encephalitis in the NY metropolitan area occurred in urban communities. The highest rate in 1999 was in Queens, NY and in 2000, Staten Is., NY. The 6 cases in NJ in 2000 were from Hudson Co. (2); Bergen Co. (1); Monmouth Co. (1); Morris Co; (1), and Passaic Co. (1). It is not possible to determine the exact locations where individuals were bitten by infectious mosquitoes. The WNV epizootic in birds in 2000 extended north to the Canadian border and south to North Carolina. The likely geographic range for WNV and the locations, which will be of highest human risk in 2001, are unknown. A major urban epidemic (453 cases) occurred in Bucharest, Romania in 1996 but was followed in 1997 with fewer (14 cases) and more rural distribution of cases. A study by NY State Dept. of Health found a correlation between the highest number of dead crows reported per square mile per week and the most human cases. Minimum Infection Rates (MIR) for each species of vector mosquito can give an estimate of increased exposure risk from mosquitoes. Some MIR for *Culex spp*. on Staten Is., NY in August, 2000 were over 15 (at least 15 infected mosquitoes per 1,000 tested).
Specific population(s) at increased risk	The elderly are at increased risk of severe disease from WNV. Those exposed to more frequent mosquito bites, especially in urban areas, are at increased risk of infection.
Quantification of exposure levels to population(s) at increased risk (i.e., susceptible sub-populations) (include indoor air as separate category as appropriate)	Specific quantification is not possible at this time; additional research on the vector competence of different mosquito species, the relationship of surveillance data to increased risk, and a better understanding of the ecology of WNV in the US is required. However, surveillance which demonstrates the presence of infected crows and vector mosquitoes in an area indicates that the population is at increased risk, and additional mosquito control intervention and personal precautions are necessary.
Dose/Impact-Response Assessment	
Quantitative dose/impact-assessment employed for each population considered	N/A
Risk Characterization	

Risk estimate(s) by population at risk including probability and number of cases/occurrences (specify risk metric employed , e.g., mean population risk upper percentile population risk, etc.)	A serosurvey in Queens, NY following the 1999 epidemic indicated that 2.6% of people in northern Queens had evidence of WNV infection. Serosurveys in 2000 found rates of 0.46% (Staten Is.), 0.12 (NYS) and 0.0 (CT). As many as 150 people may have mild or unapparent infections for every severe case. 2000 surveillance data in NJ indicates that Bergen and Middlesex Counties had the highest levels of WNV positive crows and mosquitoes in the state, and therefore may have had an increased risk of human disease (had there not been additional educational and mosquito control intervention).
Assessment of severity, persistence, irreversibility, frequency of effect(s) (categories as appropriate)	Adverse health effects usually very low severity for general population (majority asymptomatic). Fatality rates 3% to 15% of hospitalized patients, highest in elderly (only NJ fatality was elderly patient). Severe disease may include encephalitis and meningitis. Intensive supportive therapy may be required. Severe symptoms not always reversible.
Size of population(s) affected	In 1999, 0 cases of confirmed WNV in NJ. In 2000, 6 confirmed cases in NJ out of 8.4 million population.
Assessment of uncertainties in this assessment (H,M,L) and brief description, and data gaps	If there may be 150 cases of mild or inapparent infection for each confirmed case, an estimated 900 persons could have been infected with WNV in NJ in 2000. Estimated rates are from Romania and New York serosurveys.
Potential for additional data to result in a significant future change in this risk estimate (H,M,L) and brief description	Since WNV was first discovered in NA in 1999, additional data on the epidemiology and ecology of WNV, it's hosts, and vectors is required before risk estimates for NJ can be established.
Potential for future changes in the underlying risk from this stressor (+++, ++, +, 0, , ≈, where + is improvement)	Educate public to prevent exposure: ++ Improve human, avian, horse and mosquito surveillance: + Improve preventive mosquito surveillance and control: + Improve coordination of multiagency response: + Environmental factors impacting WNV cycle: 0
Potential impact from catastrophic (low probability) events (H,M,L) and brief description of likelihood	L Floodwater in July/August could increase mosquito /WNV risk.
Extent to which risks are currently reduced through in-place regulations and controls	Effective public education, surveillance and mosquito control reduce risks.
Relative Contributions of Sources to Risk (H,M,L)	
Allocation of stressor-specific risk to primary NJ sources	
Large business/industry	L
Small business industry	L

Transportation	L
Residential	M Container breeding mosquitoes
Agriculture	L
Recreation	M Exposure to mosquito bites
Resource extraction	L
Government	L
Natural sources	M Mosquito breeding sites, wild bird reservoirs of WNV
Contaminated sites	M Source of *Culex pipiens*
Diffuse and non-NJ sources	
Sediment	
Soil	
Non-local air sources (including deposition)	
Biota sinks	

Severity of specified health effects at current levels of exposure (H,M,L) (also 1-5 with 1 being least severe)	Size of population at significant risk for each health effect (H,M,L) (also 1-5 with 1 being smallest)	Are there discrete communities at elevated risk? (Y,N) (also 1-5 with 1 being the lowest probability that there are discrete communities at elevated risk)	Overall risk ranking (as a function of severity and population effected integrating across health effect) (H,M,L) (also 1-5 with 1 being the lowest overall risk)
WNV transmission L,1	L, 1	elderly Y, 1	L, 1
			L, 1

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